PB-234 920

# HEALTH CONSEQUENCES OF SULFUR OXIDES

## A REPORT FROM CHESS, 1970-1971

NATIONAL ENVIRONMENTAL RESEARCH CENTER

MAY 1974

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PB 234 920

1. REPORT NO.
EPA-650/1-74-004

2.

4. TITLE AND SUBTITLE
HEALTH CONSEQUENCES OF SULFUR OXIDES:
A Report from CHESS, 1970-1971

5. REPORT DATE
May 1974

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)

8. PERFORMING ORGANIZATION REPORT NO.

9. PERFORMING ORGANIZATION NAME AND ADDRESS
National Environmental Research Center
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, North Carolina 27711

10. PROGRAM ELEMENT NO.
1A1005

11. CONTRACT/GRANT NO.

12. SPONSORING AGENCY NAME AND ADDRESS

13. TYPE OF REPORT AND PERIOD COVERED
Final, 1970-1971

14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

16. ABSTRACT

Community Health and Environmental Surveillance Systems (CHESS) studies provide dose-response information relating short- and long-term air pollution exposures to adverse health effects. This report presents results of studies in CHESS communities in New York and the Salt Lake Basin during 1970-1971. Studies in Idaho-Montana, Chicago, and Cincinnati, employing health indicators similar to those used in CHESS, are also included. Attention is focused on effects of sulfur oxides, but the relative contribution of various pollutants, especially sulfur dioxide, particulates, and suspended sulfates, is considered. Health indicators of long-term pollution effects included acute and chronic respiratory illness and ventilatory function. Indicators of short-term effects were cardiopulmonary symptoms and asthma. Threshold estimates for the pollutants considered support existing National Primary Air Quality Standards for long-term exposures. For short-term exposures, the studies indicated adverse effects even on days below the Standards for 24-hour levels of sulfur dioxide and particulates. These effects, however, appear to be associated with suspended sulfates.

17. KEY WORDS AND DOCUMENT ANALYSIS

Acute respiratory disease
Air pollution (health effects)
Air quality standards
Asthma
Cardiopulmonary illness
CHESS (Community Health and Environmental Surveillance System)
Chronic respiratory disease
Epidemiology
Health effects (air pollution)
Particulates
Respiratory disease
Sulfur dioxide
Sulfur oxides
Suspended sulfates
Total suspended particulates
Ventilatory function

18. DISTRIBUTION STATEMENT
Release Unlimited

19. SECURITY CLASS *(This Report)*
UNCLASSIFIED

20. SECURITY CLASS *(This page)*
UNCLASSIFIED

21. NO. OF PAGES
428

22. PRICE
$5.60

EPA Form 2220-1 (9-73)

Reproduced by
NATIONAL TECHNICAL
INFORMATION SERVICE
U S Department of Commerce
Springfield VA 22151

EPA-650/1-74-004

$3.10
SN. 5500-00121

# HEALTH CONSEQUENCES OF SULFUR OXIDES:
# A Report from CHESS, 1970-1971

Human Studies Laboratory

Program Element 1A1005

1-A

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
NATIONAL ENVIRONMENTAL RESEARCH CENTER
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May 1974

## EPA REVIEW NOTICE

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# ABSTRACT

Epidemologic studies of the U.S. Environmental Protection Agency's Community Health and Environmental Surveillance System (CHESS) program provide dose-response information relating short-term and long-term pollutant exposures to adverse health effects. This report presents results of studies conducted in CHESS communities in New York and the Salt Lake Basin during 1970-1971. In addition, studies conducted in Idaho-Montana, Chicago, and Cincinnati, in which health indicators similar to those used in CHESS were employed, are included. In this report, attention is focused on the health effects associated with sulfur oxides, but the relative contribution of various air pollutants, especially sulfur dioxide, total suspended particulates, and suspended sulfates, to observed disease frequencies is considered. Health indicators of long-term pollution effects employed in these studies included increased prevalence of chronic bronchitis in adults, increased acute lower respiratory infections in children, increased acute respiratory illness in families, and subtle decreases in ventilatory function of children. Health indicators for short-term pollution effects were aggravation of cardiopulmonary symptoms and of asthma. Threshold estimates were developed for the effects of the pollutants considered. These estimates support existing National Primary Air Quality Standards for long-term exposures, insofar as these standards could be assessed in terms of the health indicators mentioned. With regard to short-term exposures, the studies indicated that adverse effects were being experienced even on days below the National Primary Standard for 24-hour levels of sulfur dioxide and total suspended particulates. These adverse health effects, however, appear to be associated with suspended sulfate levels rather than to the observed concentrations of sulfur dioxide and total suspended particulates, as evidenced by the consistency of the relationship between symptom aggravation and sulfate levels and the lack of consistency for this relationship with other pollutants.

# PREFACE

This is a report on a series of extensive epidemiologic investigations on the health consequences of sulfur oxides being carried out under the Community Health and Environmental Surveillance System (CHESS) conducted by the Environmental Protection Agency in cooperation with local and state governments, universities, and private research organizations.

The studies are presented in monograph form in order to provide the opportunity to assess the consistency of the findings from multiple studies. The health effects of pollution exposure may often be subtle rather than overt, and a single study may only suggest an adverse health effect. A series of studies showing a consistent pattern, however, will provide evidence to incriminate or absolve exposures to various levels of given pollutants as antagonistic to health. At the expense of some duplication, each study is presented in detail so that it can be read independently of the other studies.

CHESS health indicators focus on measures of selected acute and chronic disease, altered physiology, and pollutant burdens. Acute health effects attributable to short-term pollutant exposure are quantified by comparing response frequency against daily variations in pollution levels. Effects attributable to long-term exposure are identified by contrasting disease prevalence in High and Low exposure communities.

These studies describe the wide and complex range of subtle and overt adverse health effects that may be attributed to pollutant exposures but leave a number of problems unanswered that can only be partially resolved at this point. Examples of these unanswered problems include the relative health effects of sulfur dioxide and suspended sulfates, the relative importance of repeated peak short-term exposures compared to elevated annual average exposures, and the relative significance of indoor air pollutants.

Studies included in this monograph are the "state of the art" summary of our findings to date. As new techniques and areas of study appropriate to CHESS are identified, they will be incorporated into the CHESS program. Developmental work is now in progress to deploy more sensitive and objective health indicators, particularly physiologic and biochemical responses, of short-term variations in air quality. The questions raised by these present studies and the potential need for modifying existing short-term primary standards or establishing standards for other pollutants will be validated or rejected by future reports from ongoing CHESS and other studies. CHESS studies on effects attributable to oxides of sulfur, suspended sulfates, and other particulates are programmed through 1975.

Studies reported in this monograph were conducted in only a portion of the current CHESS program. Results are given for three CHESS communities in New York and four CHESS communities in the Salt Lake Basin. In addition, the monograph includes findings from studies conducted in Idaho-Montana, Chicago, and Cincinnati, in which health indicators similar to those used in CHESS were employed. Air measurements from these areas were obtained from local agencies and other Federal networks, however, and were somewhat dissimilar to standard CHESS air monitoring techniques.

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# CHAPTER 1

# INTRODUCTION

## 1.1 AN OVERVIEW OF CHESS

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## INTRODUCTION

CHESS, an acronym for the Environmental Protection Agency's (EPA) Community Health and Environmental Surveillance System, is a National program of standardized epidemiologic studies designed to measure simultaneously environmental quality and sensitive health indicators in sets of communities representing exposure gradients to common air pollutants. The purpose of the CHESS program is to evaluate existing environmental standards, obtain health intelligence for new standards, and document the health benefit of air pollution control. In order to accomplish this mission, it is necessary to provide, as far as possible, consistency in design and in methods of collecting, processing, and analyzing data among the various studies. For this reason, the program is coordinated by a single organization in cooperation with local public health agencies, universities, and private research institutes. The elements of the CHESS program are described in following sections.

## CHESS STUDY STRATEGIES

A CHESS set consists of a group of communities selected to represent an exposure gradient for designated pollutants. Each CHESS set generally includes High, Intermediate, and Low exposure communities selected to evaluate existing air quality standards for particulates, sulfur oxides, nitrogen oxides, and photochemical oxidants. Current CHESS area sets (as of March 1973) are shown in Figure 1.1.1. The possibility of detecting pollutant interactions is enhanced by selecting CHESS sets with exposure to a single pollutant alone or in combination with other pollutants. Because effects of short-term carbon monoxide exposures are more precisely studied in controlled exposure chambers, a CHESS area set was not established to measure carbon monoxide effects.

In addition to satisfying the pollutant exposure gradient, other bases for community selection within a CHESS set were that the communities be as close to one another as possible to minimize climatalogical

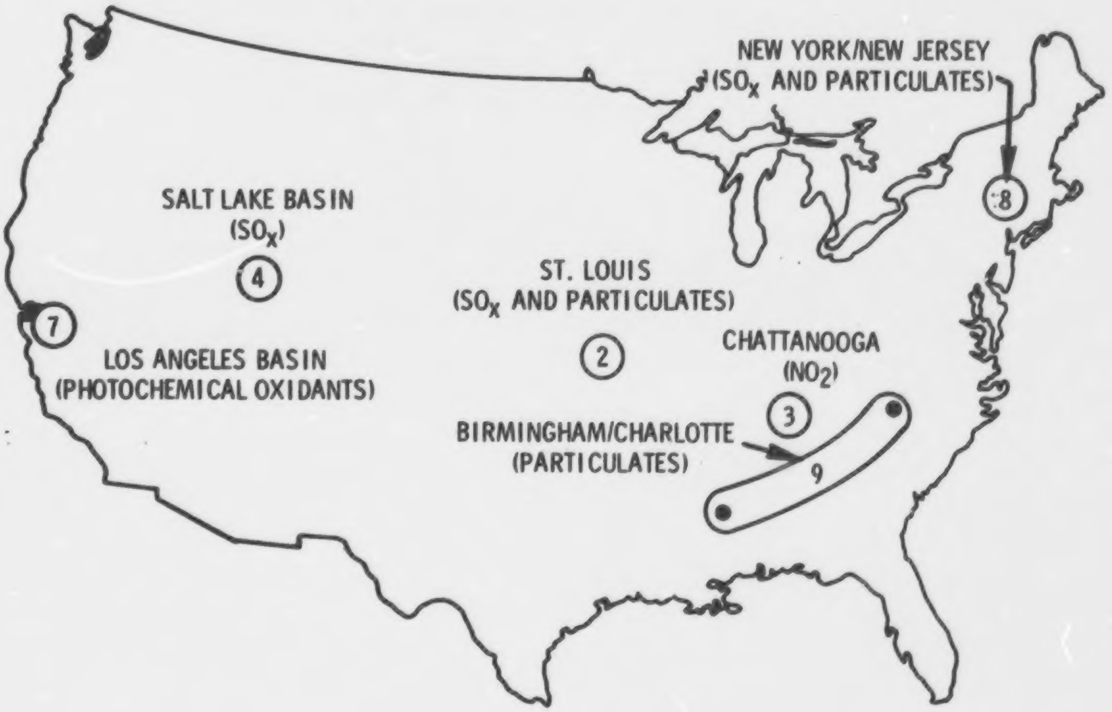

Figure 1.1.1. CHESS area sets (March 1973).

variation and be made up of predominantly white, middle-class populations that were matched, as far as possible, for socioeconomic and other demographic factors.

The samples chosen for study within a community are not intended to be representative of the general population. Air quality standards are being established to protect particularly vulnerable segments of the population, so that for some studies we deliberately recruit asthmatics and subjects with preexisting heart and lung disease. Middle-class residential neighborhoods containing three or four elementary schools and a junior and senior high school are also chosen because they represent a large proportion of the population, have a more homogeneous family and social class distribution, and represent neighborhoods whose characteristics change more slowly than those of central city neighborhoods.

CHESS programs will operate from 3 to 5 years in most areas. Measurement of sensitive health indicators during an interval of improving air quality is an optimal way to quantitate the health benefits of pollutant controls. Studies may be extended to detect time lags between air quality improvement and anticipated health benefit.

Environmental pollutants can affect the health of individuals over a broad spectrum of biological response, as shown in Figure 1.1.2. More severe effects such as death and chronic disease will be manifested in relatively small proportions of the population, and the role of environmental pollutants in the mortality and chronic disease experience of a community is difficult to quantify because so many other determinants of death and disease cannot be adequately measured. The lower strata in the response spectrum of Figure 1.1.2 are subclinical manifestations of pollutant exposure. At a point in time, many more individuals in an exposed community will respond with altered physiology or pollutant burdens (tissue residues of pollutants that accumulate in the body) than will die or develop chronic disease. Furthermore, lower levels of the response spectrum can be more readily quantified and measured objectively. Because responses at lower levels are more rapidly manifested, they may be more useful to demonstrate an immediate health benefit of pollution control.

CHESS health indicators focus on measures of selected acute and chronic diseases, altered physiology, and pollutant burdens. These indicators may be conveniently divided into two categories: health effects attributable to short-term pollutant exposure and effects attributed to long-term exposure. Sample sizes and response frequencies employed for each CHESS health indicator are shown in Table 1.1.1. Acute health effects are observed by following panels of subjects who have been systematically preenrolled and comparing response frequency against daily variations in pollutant levels. Various statistical analyses are employed to isolate the effects of temperature and season on response frequency. By making simultaneous observation in Low exposure communities, effects of environmental covariates such as temperature can be quantified, and pollutant-temperature interactions can be detected. These health indicators are related to exposures of 1 to 96 hours, depending upon the nature of the response.

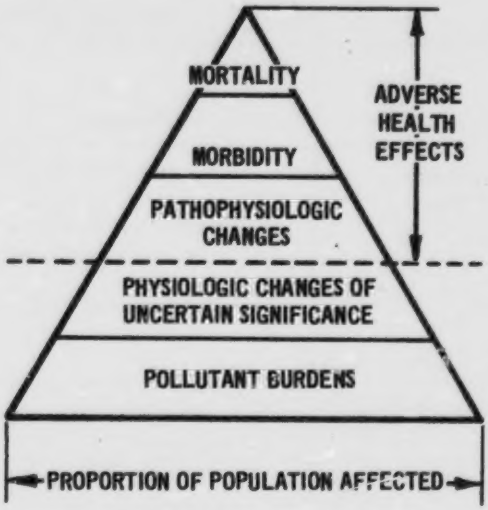

Figure 1.1.2. Spectrum of biological response to pollutant exposure.

Effects attributable to long-term exposure are identified by contrasting disease prevalence in High and Low exposure communities. An acute effect, such as excess acute respiratory disease, may be a manifestation of chronically impaired resistance to disease. Persistence of illness excess or of altered physiology in a High exposure community provides a means to discriminate between effects attributable to short-term and to long-term exposure. Disentangling the effects of dose rate, that is, large doses in short intervals versus repeated small doses over long periods, is difficult in community studies and generally requires controlled experimentation.

Table 1.1.1. CHESS HEALTH INDICATORS

| Exposure | Indicator | Sample size per CHESS community | Frequency of response |
|---|---|---|---|
| Short-term ($<$4 days) | Frequency of asthma attacks | 50 to 75 | Daily |
| | Aggravation of chronic respiratory symptoms | 50 to 75 | Daily |
| | Aggravation of cardiac symptons | 50 to 75 | Daily |
| | Acute irritation symptoms during episodes | 400 to 1000 | 3 to 4 times yearly |
| | Daily mortality | Variable | Daily |
| Long-term (usually $\geq$1 year) | Pollutant burdens | 800 to 1200 | Once in 2 years |
| | Impairment of lung function | 1500 to 2000 | 3 times yearly |
| | Incidence of acute respiratory disease in families | 1000 | Once every 2 weeks |
| | Frequency of acute lower respiratory disease in children | 1500 to 3000 | Once in 2 years |
| | Prevalence of chronic respiratory disease | 1500 to 3000 | Once in 2 years |

Many factors contribute to community differences in the distribution of diseases associated with air pollution exposure. Factors that, along with environmental pollution, codetermine the quantitative level of a health indicator in a community are called "covariates." Covariate information obtained in the CHESS program includes data on other environmental factors, such as temperature and humidity, and personal or family characteristics, such as age, sex, race, education of parents, occupational dust and fume exposure, cigarette smoking habits, geographical migration, and previous illness experience. Covariates may be kept constant across study groups by careful selection of participants, or they may be measured and then appropriately adjusted through application of statistical procedures.

## ENVIRONMENTAL MONITORING

Air monitoring stations are sited in each CHESS community to provide credible estimates of pollutant exposure for the study population. The large majority of study subjects live within 1.5 miles of the monitoring station. Stations and sample inlets are placed to be as representative of community-wide exposure as possible. Topography, land use adjacent to the station, emission sources, and population distribution are considered in selection of CHESS monitoring sites.

At the inception of the CHESS program in 1969, manual instruments were operated 7 days each week to monitor 24-hour integrated concentrations of total suspended particulates, suspended sulfates, suspended nitrates, sulfur dioxide, and nitrogen dioxide; monthly concentrations of dustfall and trace metals in dustfall were also determined. Since that time, instrumentation has been added to measure respirable suspended particulates, carbon monoxide, ozone, and total hydrocarbons, and automated or refined analysis procedures have been incorporated in the system. Beginning in January 1972, automated sampling methods listed in Table 1.1.2 were introduced into some CHESS stations, and the entire monitoring

**Table 1.1.2. CHESS ENVIRONMENTAL MONITORING**

| Measurement device[a] | Type of measurement | Sampling period | Pollutant | Analysis method | Period employed |
|---|---|---|---|---|---|
| High-volume sampler | Manual | 24 hours | Total suspended particulate | Gravimetric | Since 1969 |
| | | | Suspended sulfates | Turbidimetric<br>Methylthymol blue | 1969-1971<br>Since 1971 |
| | | | Suspended nitrates | Hydrazine sulfate-copper sulfate<br>Copper-cadmium | 1969-1971<br>Since 1971 |
| | | | Trace metals | Atomic absorption | Since 1969 |
| Cyclone | Manual | 24 hours | Respirable suspended particulate (<5 μm) | Gravimetric | 1972-1973 |
| Cascade impactor | Manual | 24 hours | Total suspended particulate | Gravimetric | Since 1970 |
| | | | Respirable suspended particulate | Gravimetric | Since 1970 |
| Tape sampler | Automated | 2 hours | Suspended particulates | Optical density | 1970-1972 |
| Dustfall bucket | Manual | Monthly | Settleable particulates | Gravimetric | Since 1970 |
| | | | Trace metals | Atomic absorption | Since 1969 |
| Bubbler train (TCM) | Manual | 24 hours | Sulfur dioxide | West-Gaeke | Since 1969 |
| Autoanalyzer | Automated | Continuous | Sulfur dioxide | Flame-photometric | Since 1974 |
| Bubbler train (sodium hydroxide) | Manual | 24 hours | Nitrogen dioxide | Jacobs-Hochheiser | Since 1969 |
| Autoanalyzer | Automated | Continuous | Nitrogen dioxide | Chemiluminescent | Since 1974 |
| Autoanalyzer | Automated | Continuous | Carbon monoxide | Nondispersive infrared | Since 1974 |
| Autoanalyzer | Automated | Continuous | Ozone | Chemiluminescent | Since 1974 |
| Autoanalyzer | Automated | Continuous | Total hydrocarbons | Flame emission | Since 1974 |
| Autoanalyzers | Automated | Continuous | Temperature, humidty, wind speed, and wind direction | Weather Bureau standard | Since 1974 |

[a] Not all measurement devices are employed at each CHESS site.

system is now being automated. Automated stations will telemeter real-time pollutant measurements to a central processing station, providing data to relate short-term environmental variations to health indicators of acute response. Further details on measurement methods and instrumentation are given elsewhere in this monograph.[1]

Reagents and filters used in manual instruments are prepared centrally and periodically checked at designated quality control points before shipment to field stations. Instruments are also calibrated and maintained centrally. Environmental samples obtained in the field are processed at a central laboratory in which systematic checks of procedures are made. Duplicate side-by-side monitors are operated intermittently at selected field stations to determine reproducibility of individual sampling trains. Automated instruments, having on-call telemetric output, permit daily routine instrument performance checks from the central processing point. Computer programs are presently being developed to monitor daily output of automated instruments and to flag deviations from expected performance. CHESS quality control features are more fully described elsewhere.[1]

Stationary monitors have inherent drawbacks as estimators of human exposure. Within short-term frames, individuals frequently change between indoor and outdoor environments, from one neighborhood to another, and from residential to occupational exposures. Quiet, acceptable, small-scale instruments for personal monitoring have not been developed, though the need has been evident for years. Likewise, quiet indoor air monitors having desirable response characteristics have yet to be marketed. These deficiencies require reliance on stationary monitors to estimate current environmental exposures of communities.

Attempts to relate chronic disease to long-term exposures are fraught with methodologic difficulties. Chronic disease is likely to result from cumulative exposures of several years to several decades. Quantitative air monitoring data for a given neighborhood or city usually are available only for the past few years. Attributing area differences in chronic disease prevalence to current pollutant exposures frequently will lead to underestimates of the true exposure associated with illness. During the 1940's and 1950's, when coal was widely consumed for domestic heating and industrial control measures were less prevalent, air pollutant concentrations tended to be considerably higher than values measured in 1970 or 1971. This is especially true in cities where stationary sources are principal contributors to community air pollution. Therefore, in order to assess accurately the magnitude of the dose-response relationship, estimates of past exposure are required and have been made in CHESS chronic disease studies. In communities dominated by a single point source, these estimates are based on past emissions. Emissions data and meteorologic information are entered into a meteorologic dispersion model, yielding estimates of ambient exposure. These results are calibrated to current ambient air monitoring data. In multiple-source urban areas, adequate past emissions data seldom are available, and dispersion models tend to cope inadequately with the complex variability of urban exposures. In these situations, local suspended particulate or dustfall measurements dating back to the early 1950's, when available, proved to be a crude but necessary baseline for extrapolating from current air monitoring data to past exposure.

In addition to long-term exposure estimates, some CHESS studies attempt to discriminate between chronic disease prevalence of long-time residents and recent immigrants to a High exposure area. After discounting the relatively high illness rates of most recent immigrants (residence duration of 1 year or less), disease prevalence is compared in residents of 2 to 4 years duration from High and Low exposure areas. Other things being equal, excess disease found among recent residents of High exposure areas may be attributed to more recent exposure. If the effect is cumulative, area differences in disease prevalence will tend to be amplified among long-term residents and will confirm the findings among recent immigrants. These approaches provide one means to assess the impact of exposure duration on chronic disease prevalence.

## DATA ANALYSIS

Since sample sizes for community studies necessarily are large, the computer is an essential part of the data processing operation. Not only does it perform the standard storage and retrieval function for health and environmental exposure data and perform statistical analyses, but it also aids extensively in the study logistics. The computer is used to select candidates in priority order for the repetitive panel studies, to print required identifying information on the questionnaires, and to prepare mailing labels for health collection materials. To lessen an enormous amount of data coding and keypunching, optically sensed questionnaire forms are used. Although the information on these forms is read directly onto magnetic tapes, much data editing is still necessary. For each type of questionnaire, edit computer programs are utilized to identify errors.

As with the environmental monitoring samples, all questionnaire development, data processing, and statistical analysis are performed at a central facility; hence, uniform control of these important functions is maintained.

Statistical analyses of CHESS data sets require several different techniques. Standard analyses are not always possible since the dependent health variables are confounded by discrete design variables and continuous covariates. These analyses had to allow for unbalanced designs, missing data, repeated measurements, serial correlation, and nonnormality. Most of the quantitative health variables, such as pulmonary function tests, were analyzed using a general linear regression model as described by Graybill.[2] These models allow for both continuous and discrete covariates and do not assume that these covariates are linear (the word "linear" in linear model, refers only to the coefficients of these effects). Most of the categorical responses, such as chronic respiratory or acute lower respiratory disease responses, were analyzed using a general linear model, as described by Grizzle et al.[3] This model is also a least squares procedure and is very similar to the regression model. This procedure produces marginal means adjusted for the other factors or covariates and partitions Chi Squares in a manner similar to the partitioning of sums of squares in regression. The procedure is robust (i.e., moderate deviations from underlying assumptions will not alter conclusions about the statistical significance of observed differences), except for small cell sizes. In most analyses, small cell sizes were not a problem. These analyses permit testing and estimating of area differences after accounting for the previously mentioned covariates.

## SUMMARY

This paper has described the structure and rationale of the CHESS program, the methods employed to relate environmental exposure to human health effects, and the various study strategies used to implement this program. Subsequent papers in this monograph will present detailed exposure-effects information derived from the CHESS program in its first full year of operation. Papers will focus on effects related to sulfur oxide exposures.

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# CHAPTER 2
# SALT LAKE BASIN STUDIES

## 2.1 HUMAN EXPOSURE TO AIR POLLUTANTS IN SALT LAKE BASIN COMMUNITIES, 1940-1971

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## INTRODUCTION

The Salt Lake Valley offers a unique setting in which to observe the relationships between the health parameters of the Community Health and Environmental Surveillance System (CHESS) program and a gradient of human exposure to sulfur dioxide. Previous epidemiological studies investigating sulfur dioxide effects have been confounded by mixed exposures in urban areas to total suspended particulates and to other pollutants. Four communities in or near the Salt Lake Valley were chosen to represent an exposure gradient to sulfur dioxide, which is emitted primarily from a large copper smelter west of the study communities. It was also expected that the exposure levels of the communities to other pollutants would be below the National Ambient Air Quality Standards and that concentrations of suspended particulates in the communities would be similar.

Emission controls have already been added to the copper smelter near the Utah CHESS communities, and further controls are planned. In fact, the primary sulfur dioxide emission from the smelter in 1971 was less than one-fifth the emission of 1940.[1] Thus, the Utah CHESS study allows health effects to be observed as the level of sulfur dioxide decreases.

The purpose of this report is to provide long-range pollutant exposure information that can be used for studies of human health effects within these Utah communities. These health effects studies relate both chronic and acute respiratory diseases and other health effect indicators to human air pollution exposures.

### Community Descriptions

The Utah CHESS study areas are situated in the north central part of the state of Utah and include Ogden, Salt Lake City, Kearns, and Magna (Figure 2.1.1). These communities were selected on the basis of meteorological information, emissions inventories, and previous air quality measurements to provide an exposure gradient for sulfur dioxide with similar levels of other pollutants, which were expected to be below National Ambient Air Quality Standards. All four communities have primarily white, middle-class populations.

Except for Ogden, the study areas are located on the floor of the Salt Lake Valley, 4220 feet above mean sea level (Figure 2.1.1). Great Salt Lake lies to the northwest of the communities in the valley, the nearest point of the lake being about 12 miles from Salt Lake City. The ridges of the Wasatch Mountains, which rise to a height of 11,300 feet, and the ridges of the Oquirrh range, which rise to a height of 10,000 feet, form a formidable barrier to east-west movement of air masses.

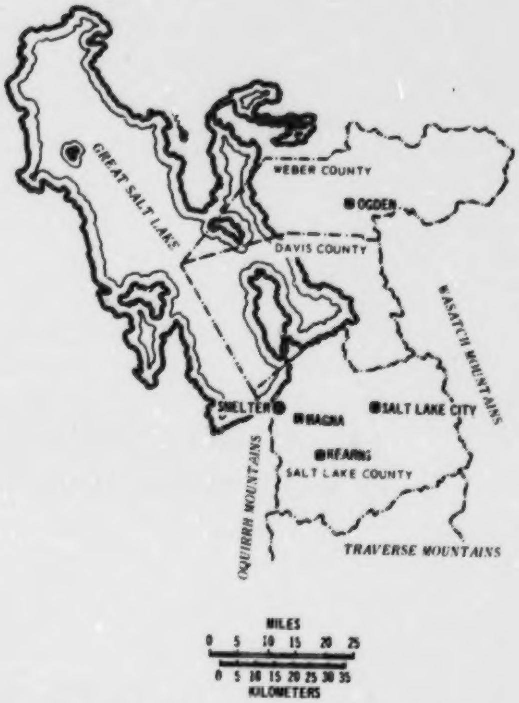

Figure 2.1.1. Salt Lake Valley CHESS communities.

Because of the topographic features of the area, local climatology plays an important role in air pollution considerations. In particular, wind flow around Salt Lake City, for the most part, is a function of the proximity of the Salt Lake Valley to the canyons on the west slopes of the Wasatch Mountains. Differential heating by the sun results in up-slope winds in the afternoon and evening and down-slope winds at night and in the morning.

An inspection of the wind rose for the Salt Lake City Airport for 1951-1960 (Figure 2.1.2) reveals that about 50 percent of the winds blow from the south to southeast and 25 percent from west-

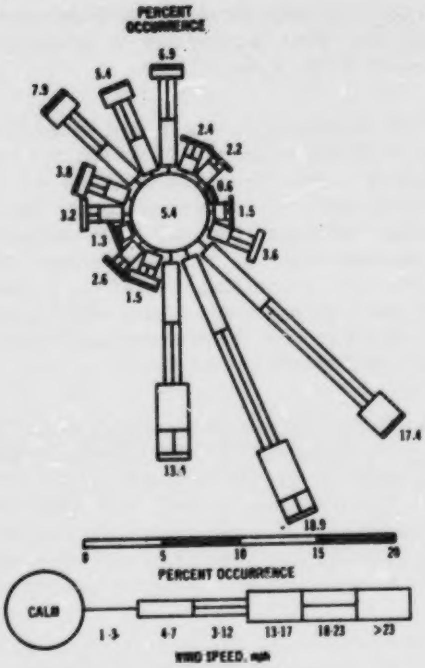

Figure 2.1.2. Surface climatologic wind rose, Salt Lake City Airport.

northwest to north, there are 5 percent calms, and the remaining winds (20 percent) are spread around the other directions. Winds from the south to southeast represent nighttime down-slope winds, which usually persist about 15 hours, 9 p.m. to 12 noon, and result from cool air flowing down the slopes of the Salt Lake Valley to the south and the Wasatch canyons to the east and southeast of Salt Lake City. With daylight, heating of the mountain slopes causes an up-valley flow (WNW-N) to start about noon and continue during the afternoon and evening. Mean velocity of the up-valley winds is about 9 mph, and that of the down-valley winds is about 7 mph.

A mountain-valley wind pattern such as this has important implications for the long-term air pollution situation around Salt Lake City. Pollutant sources to the north and northwest or those to the south and southeast would affect the city about 75 percent of the time. There are two major oil refineries to the northwest, both possible sources of sulfur dioxide, and it is reasonable to expect that emissions from both sources would flow directly into Salt Lake City with the winds that persist from noon to 9 p.m. almost every day. However, the greatest source of sulfur dioxide around Salt Lake City and the CHESS areas is the copper smelter near Garfield, Utah. This large point source lies 13 miles due west of the city, and from that location winds that would carry emissions directly to the city would occur only 4 percent of the time.

Two CHESS communities, Magna and Kearns, are located much closer to the smelter source, approximately 5 miles and 8 miles from the smelter, respectively, on the floor of the Salt Lake Valley between Salt Lake City and the smelter. It was thought these sites would receive a high exposure of sulfur dioxide with the up-valley (WNW-N) winds in the Salt Lake Valley.

The remaining CHESS site, Ogden, lies out of the Salt Lake Valley 35 miles due north of Salt Lake City in the foothills of the Wasatch Mountains. Inspection of summarized wind data for Ogden (Figure 2.1.3) reveals a pattern different from that around Salt Lake City. Down-slope winds out of Weber Canyon and

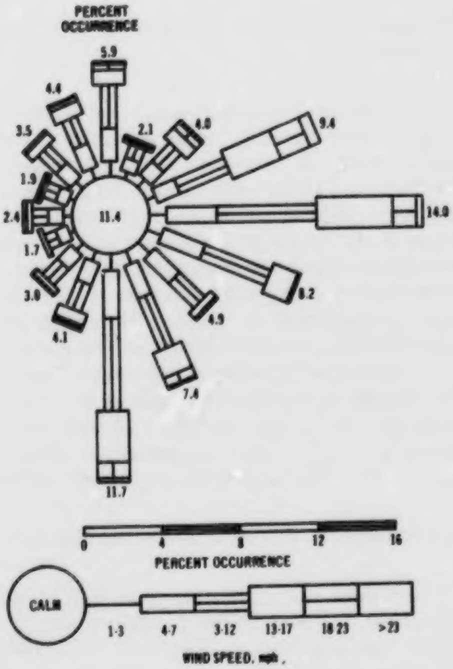

Figure 2.1.3. Surface climatologic wind rose, Ogden (Hill Air Force Base).

other large canyons to the east result in prevailing easterly winds from the Wasatch Mountains. A southerly flow, which might carry sulfur dioxide emissions from Salt Lake City, is evident.

Climatological data from the radiosonde station at Salt Lake City Airport rank this region as one of the poorest in the nation for total hours of inversion conditions and also for total number of days during which inversion occurs. Inversions are observed on about 80 percent of the mornings throughout the year. The highest frequency of morning inversions occurs in summer, with a frequency of from 90 to 95 percent. Although winter inversions average only 75 to 80 percent in the morning, they tend to persist during the day, particularly with stagnant conditions. In warmer months, rapid heating tends to dissipate the more frequent morning inversions. Computation of morning urban mixing depths reveals no monthly average above 400 meters; the critical morning mixing depth for high air pollution advisories is 500 meters. Another criterion for high air pollution is average wind speeds aloft of 4 m/sec through the urban mixing depth, and Salt Lake City averages less than that in 6 of the 12 months of the year.

## Monitoring Human Exposure to Air Pollutants

The strategy and methods employed in the CHESS program for monitoring human exposure to environmental pollutants have been described elsewhere.[2,3] For the Utah CHESS communities, the air monitoring sites were located within an approximate 1.5-mile radius of the center of the human population under surveillance. These air monitoring sites were located to represent the air quality exposure of the respective study populations in residential neighborhoods. Sulfur dioxide, nitrogen dioxide, and total suspended particulate concentrations, coefficient of haze levels, and dustfall have been measured at the sites since December 1970. Data on sulfur dioxide and total suspended particulate concentrations were also available from the Utah State Department of Health.

### *Location and Description of the Monitoring Sites*

The CHESS air monitoring sensors in Magna are located on the roof of the Brockbank Junior High School. The school is one-story with a roof about 20 feet above ground. There are no tall trees or other obstructions to airflow within several hundred yards of the school. The area around the school building is a grass-covered yard with a small asphalt parking lot. The school is located in the residential neighborhood. A moderately busy thoroughfare is located two blocks south of the school.

The air monitoring sensors for the Kearns community are located on the roof of the Kearns Junior High School, a one-story building except for the auditorium, which extends slightly higher. The air monitoring equipment is located at the one-story height approximately 20 feet above ground. The immediate area surrounding the school is grass-covered except for a parking lot north of the school. The school is located in the residential neighborhood with no busy thoroughfares nearby.

In Salt Lake City, the air monitoring sensors are on top of the two-story City-County Health Complex approximately 35 feet above ground. To the north and east of the building are fairly busy commercial streets. Parking lots are located to the south and west of the building. The buildings near the Health Complex are one story, and no tall trees are present.

Air monitoring sensors in Ogden are located on the roof of the one-story City-County Health Department. The roof is about 15 feet above ground. A 50-foot-tall tree is located approximately 40 feet east of the building. A fire department building to the east of the building has a smoke stack approximately 40 feet high about 200 feet from the monitoring equipment. The stack emits from the gas-fired boiler in the fire department. The streets on two sides of the building are busy commercial thoroughfares.

### *Pollutant Measurement Methods (CHESS)*

*Sulfur Dioxide* - The 24-hour sulfur dioxide gas bubbler system is composed of relatively simple parts. From an ambient air inlet, air is drawn through a 6-inch-long bubbler stem of 6-mm tubing. The tubing is drawn to an inner tip diameter of 0.025 inch to control bubble size. The air then bubbles through 35 ml of a 0.1-*M* potassium tetrachloromercurate (TCM) solution contained in a 164- by 32-mm polypropylene tube. Exhaust air passes through a glass wool moisture trap. The air then passes through a 23-gauge hypodermic needle used as a critical orifice to control the flow rate at approximately 0.5 liter/min. A second moisture trap is placed between the needle and the vacuum source. Moisture traps are used to reduce corrosion for both the vacuum pump and the needle. The traps also provide a container for overflow of TCM solution if the train is assembled incorrectly. Flow rate is measured at the beginning and end of each sample period. Chemical analysis is performed using the modified West-Gaeke procedure.[4-6] Results are reported in micrograms per cubic meter ($\mu g/m^3$).

*Nitrogen Dioxide* - Nitrogen dioxide concentrations were determined from 24-hour gas bubbler samples using the Jacobs-Hochheiser[7] technique. The nitrogen dioxide data are being reported in this paper although the method has recently come under much criticism. It is widely held that the Jacobs-Hochheiser method does not give accurate results because of several factors. Two factors include, first of all, a fluctuating absorption efficiency and, secondly, probable interference from other environmental pollutants. Although at the time of data collection the Jacobs-Hochheiser method was the Federally accepted method of measuring nitrogen dioxide concentration, these data should only be used in the light of the recent findings mentioned previously.

The 24-hour nitrogen dioxide gas bubbler system is very similar to the sulfur dioxide bubbler. The air bubbles through two 164- by 32-mm polypropylene tubes, each containing 35 ml of 0.1 $N$ sodium hydroxide. Otherwise, components and operation are the same as described above for the sulfur dioxide bubblers.

*Total Suspended Particulate* - The high-volume air sampler is used to collect fairly large quantities of particulate matter on filters. The sampler passes ambient air through an 8- by 10-inch glass fiber filter at a starting flow rate of approximately 60 $ft^3/min$. The flow rate is sufficiently large to allow collection of suspended particulates between 90 and 0.1 $\mu m$ in diameter.

The sampler and rotameter are routinely calibrated as a unit for accurate flow rate determinations. Each unit is routinely inspected and repaired every 25 calendar days. A shelter of standard design protects the sampler from adverse weather conditions and vandalism. The shelter is located sufficiently high above ground to provide unobstructed airflow.

The total weight of particulate matter collected on each filter is determined gravimetrically following the Environmental Protection Agency standard reference method.[8,9]

For the sulfate fraction of suspended particulate, a turbidimetric method is used, and turbidity is measured using a spectrophotometer.[10] The nitrate fraction is determined using a reduction diazo coupling method with automated analysis.[10] Results are reported in micrograms per cubic meter ($\mu g/m^3$).

*Dustfall Particulate* - The measurement of atmospheric particulate dustfall provides an approximate gauge of the amount of environmental contaminants that precipitate out of the atmosphere onto the respective CHESS neighborhoods. Specifically, determinations are made of total dustfall and of lead, zinc, and cadmium in dustfall. These data are useful for estimating the proportions of pollutants entering via the respiratory and gastrointestinal systems.

The dustfall unit is an open-top, cylindrical container, which collects the larger and more dense fractions of atmospheric particulates. Made of durable, nonreactive polyethylene, the container is supplied with a press-on lid. The entire unit is therefore suitable for shipment. When in use, the bucket is suspended on an aluminum mast at a height at least 6 feet above ground for a 1-month period. The particles collected are emptied into a glass beaker, water is evaporated, and the weight of the remaining matter is determined.[11] Residue is also analyzed for trace metals by atomic absorption.[12] Results are reported in grams or milligrams per square meter per month ($g/m^2/mo$).

*Coefficient of Haze (COH)* - The A.I.S.I. Tape Sampler is designed to provide continuous short-term measurements of particulate matter. The principal components of the tape sampler are a vacuum pump, sampling nozzle, automatic timer, and sampling tape (filter). The vacuum pump draws ambient air through a filter held in the sampling nozzle. Particulates are deposited upon the tape for a period of 2 hours, then the tape is automatically advanced to the next clean area. The sampler airflow, which is measured by a built-in flowmeter, is approximately 0.25 $ft^3/min$.

The unit is housed in a metal carrying case, which both protects the instrument and prevents soiling of the tape outside the sample spot. The light transmittance of each sampled area is determined at a central laboratory. Specific methods for the determination of COH units have been previously described.[13] Results are reported as COHs per 1000 linear feet of air (COHs/1000 lin ft).

*Precision of Measurements* - The CHESS program conducted tests to determine the precision of environmental measurements.[3] These tests were done mainly in the southeastern CHESS cities, Birmingham, Charlotte, and Greensboro. Duplicate sensors were operated at air monitoring sites within these cities on a daily basis for 8 months in Birmingham and for 6 months in Charlotte and Greensboro. Comparisons of the regular samples with duplicate samples were used to statistically determine the precision of CHESS measurements for sulfur dioxide, nitrogen dioxide, total suspended particulate, and particulate dustfall. The arithmetic mean percentage errors were as follows:

1. Sulfur dioxide, ±27 percent.
2. Nitrogen dioxide, ±16 percent.
3. Total suspended particulate, ±6 percent.
4. Particulate dustfall, ±25 percent.

The uncertainty associated with the nitrogen dioxide determinations using the Jacobs-Hochheiser technique should be noted once again. As mentioned in the section on nitrogen dioxide measurement method, the data should only be considered in the light of the recent findings concerning the questionable validity of the method.

*Sampling Times and Frequency* - Table 2.1.1 compares the measurement methods, sampling times, and sampling frequencies used by CHESS and the Utah State Division of Health. CHESS measurements, except for particulate dustfall and COHs, are taken daily for each 24-hour period. The 24-hour period normally begins and ends between 8 a.m. and 12 noon. The COH measurements provide a 2-hour value representing the even hours of the day, for example, 8 to 10 a.m., 10 a.m. to 12 noon, etc. Particulate dustfall measurements are taken over a 1-month period.

Table 2.1.1. COMPARISON OF CHESS AND UTAH STATE DIVISION OF HEALTH METHODS, SAMPLING TIMES, AND SAMPLING FREQUENCIES

| Measurement by | Pollutant | Method | Sampling frequency | Sampling time |
|---|---|---|---|---|
| CHESS | Sulfur dioxide | Bubbler (24-hour sample; West-Gaeke analysis) | Daily | 24 hours |
| | Nitrogen dioxide | Bubbler (24-hour sample; Jacobs - Hochheiser analysis) | Daily | 24 hours |
| | Total suspended particulate | High-volume sampler | Daily | 24 hours |
| | Total suspended particulate | A.I.S.I. Tape Sampler | Every 2 hours | 2 hours |
| | Dustfall particulates | Dustfall bucket | Monthly | 1 month |
| Utah State Division of Health | Sulfur oxides | Automatic conductivity | Continuously | 24 hours (1963-69)<br>1/2 hour (1970-71) |
| | Total suspended particulate | High-volume sampler | Daily | 24 hours |

### *Pollutant Measurement Methods (Utah)*

The Utah State Division of Health measures sulfur oxides on a continuous basis using the automatic conductivity method with hydrogen peroxide as the absorbing solution.[14] If acidic gases are present in the ambient air sample, they can be expected to interfere with this method. These measurements are summarized into 24-hour periods representing daily values for the years 1963-1969. Half-hour averages are available for 1970-1971. Total suspended particulate concentrations are determined using a high-volume air sampler similar to that used by CHESS.

## RESULTS AND DISCUSSION

### Annual Concentrations

The Utah State Division of Health has monitored suspended particulate in Salt Lake City, Ogden, Magna, and Kearns since 1963, 1965, 1968, and 1971, respectively (Figure 2.1.4). The annual geometric mean decreased between 1963 and 1967 for Salt Lake City and between 1965 and 1966 for Ogden. Beginning in 1966, however, the annual geometric means for Salt Lake City, Magna, and Ogden appear to be relatively stable. Since only the 1971 annual mean for Kearns is known, no trend can be determined.

Sulfur oxide concentrations have been determined for Salt Lake City and Ogden since 1965 and for Magna and Kearns since 1970 and 1971, respectively (Figure 2.1.5). The annual arithmetic mean for Salt Lake City dropped considerably in 1967. Between 1967 and 1971 the means increased yearly. However, the annual mean for 1971 was still considerably less than the 1965 mean. The means for Ogden fluctuated slightly between 1965 and 1970. The mean for 1971, however, showed a 4-fold increase over the value for 1970. Magna showed a slight decrease between 1970 and 1971, although no trend could be determined.

Annual averages based on the Utah State Division of Health data are tabulated in the Appendix, Tables 2.1.A.1 and 2.1.A.2.

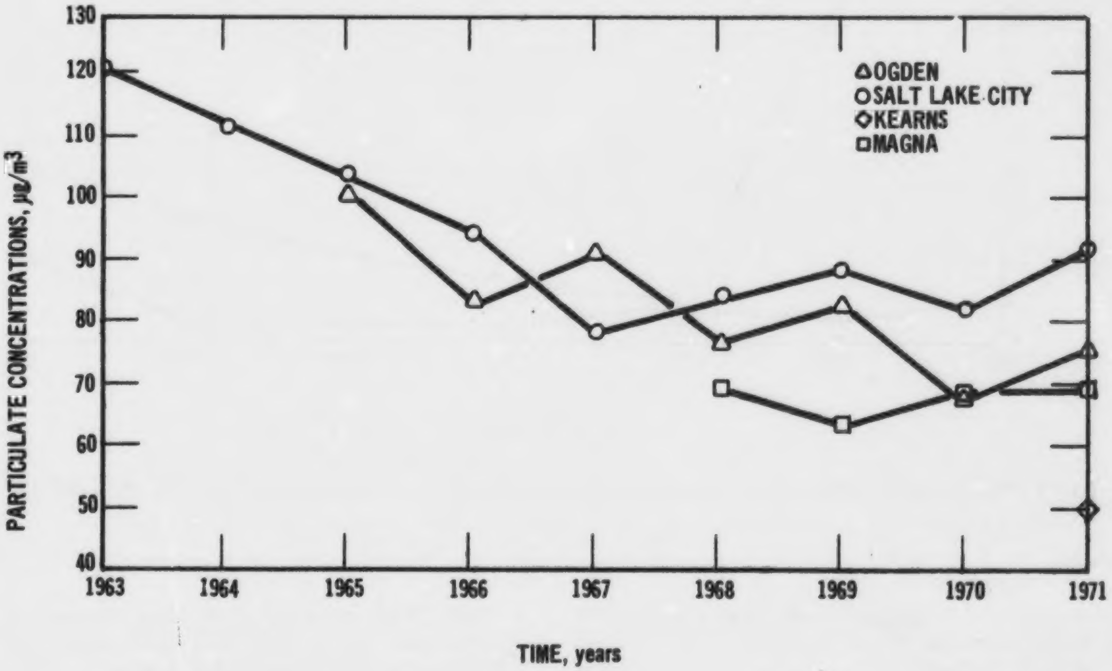

Figure 2.1.4. Total suspended particulate concentrations versus time, Utah State Division of Health, 1963-1971.

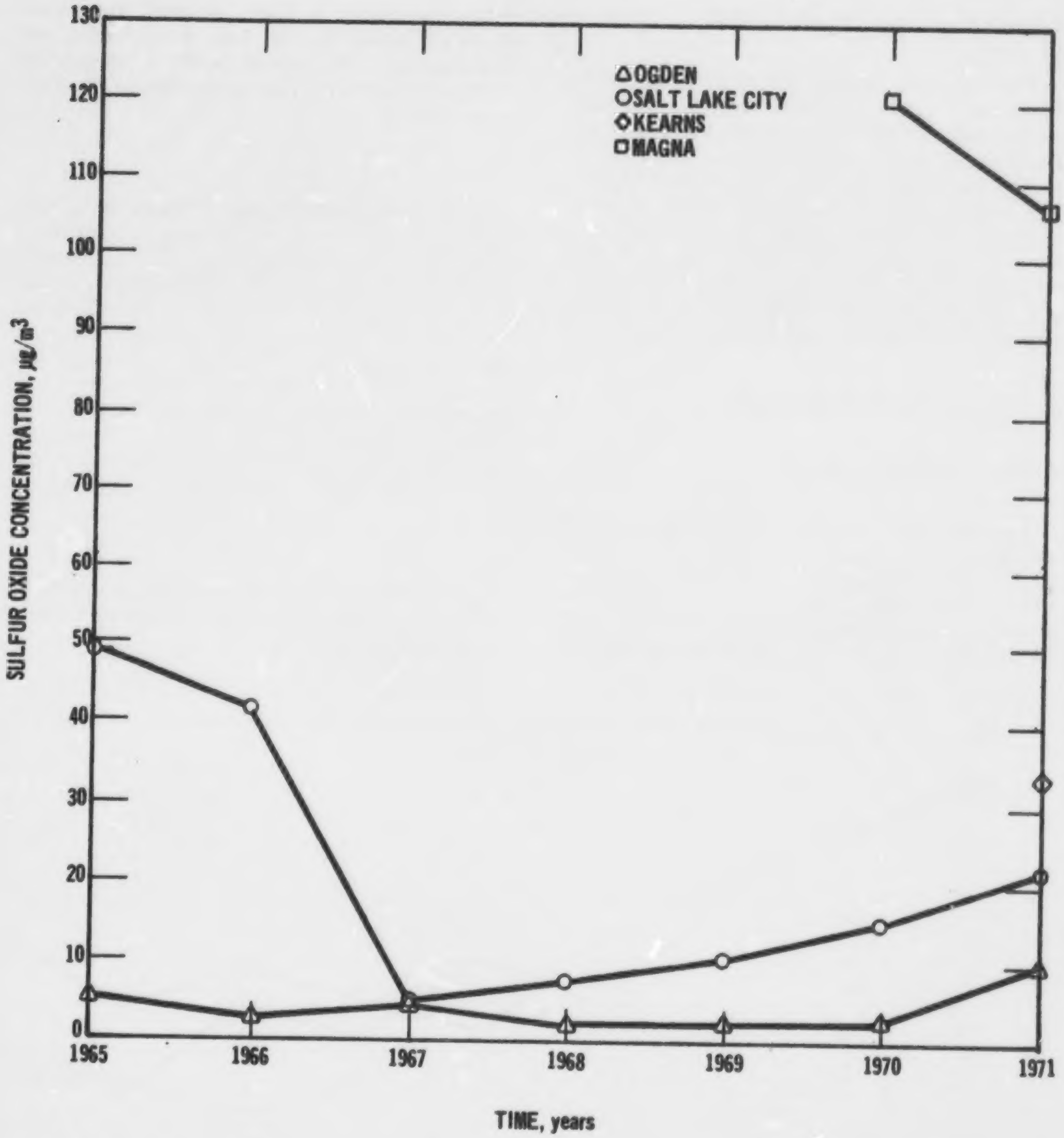

Figure 2.1.5. Sulfur oxide concentrations versus time, Utah State Division of Health, 1966-1971.

Table 2.1.2 summarizes the Utah CHESS 1971 annual averages for all pollutants. The National Primary Ambient Air Quality Standards are shown for comparison. In no case are these standards exceeded.

Table 2.1.3 compares Utah State Division of Health results with Utah CHESS results. Since Utah CHESS measurements were begun in December 1970, only the 1971 annual averages for each area are shown. For both total suspended particulate and

### Table 2.1.2. SUMMARY OF UTAH CHESS 1971 ANNUAL AVERAGES FOR POLLUTANT CONCENTRATIONS[a]

| Pollutant | Ogden | Salt Lake City | Kearns | Magna | Standard[b] |
|---|---|---|---|---|---|
| Sulfur dioxide, $\mu g/m^3$ | 8.0 | 15.3 | 21.7 | 61.8 | 80.0 |
| Suspended sulfate,[c] $\mu g/m^3$ | 4.8 | 6.1 | 6.4 | 9.6 | – |
| Total suspended particulate,[c] $\mu g/m^3$ | 66.5 | 70.9 | 38.2 | 53.9 | 75.0 |
| Nitrogen dioxide, $\mu g/m^3$ | 37.4 | 40.6 | 22.3 | 20.8 | 100.0 |
| Suspended nitrate,[c] $\mu g/m^3$ | 2.0 | 2.4 | 1.7 | 1.3 | – |
| Particulate, COHs/1000 lin ft | 0.3989 | 0.5654 | 0.2026 | 0.2776 | – |
| Dustfall particulate, $g/m^2/mo$ | 4.34 | 3.56 | 2.44 | 4.61 | – |
| Cadmium in dustfall, $mg/m^2/mo$ | 0.08 | 0.11 | 0.17 | 0.11 | – |
| Lead in dustfall, $mg/m^2/mo$ | 6.70 | 8.21 | 4.10 | 3.99 | – |
| Zinc in dustfall, $mg/m^2/mo$ | 4.26 | 3.88 | 3.68 | 6.02 | – |

[a] Concentrations are annual arithmetic mean values unless otherwise noted.

[b] National Primary Ambient Air Quality Standard.

[c] Annual geometric mean values.

### Table 2.1.3. COMPARISON OF UTAH STATE DIVISION OF HEALTH DATA WITH UTAH CHESS DATA, 1971 ANNUAL AVERAGE POLLUTANT CONCENTRATIONS

| Pollutant | Measured by | Ogden | Salt Lake City | Kearns | Magna |
|---|---|---|---|---|---|
| Total suspended particulate,[a] $\mu g/m^3$ | CHESS | 66.5 | 70.9 | 38.2 | 53.9 |
|  | Utah | 78.0 | 94.0 | 51.0 | 71.0 |
| Sulfur dioxide,[b] $\mu g/m^3$ | CHESS | 8.0 | 15.3 | 21.7 | 61.8 |
| Sulfur oxides,[b] $\mu g/m^3$ | Utah | 10.5 | 21.0 | 34.1 | 107.4 |

[a] Annual geometric mean.

[b] Annual arithmetic mean.

sulfur dioxide (sulfur oxides), the Utah CHESS and the Utah State Division of Health results showed the same pollutant gradients across the four communities, but sulfur oxide levels from the State of Utah were considerably higher than those obtained by Utah CHESS. As mentioned previously, The Division of Health uses an automatic conductivity method that measures sulfur oxides and is subject to interference, whereas the West-Gaeke method employed by CHESS is specific for sulfur dioxide.

### Daily Pollutant Cumulative Frequency Distribution

Frequency distributions and summaries of daily pollutant concentrations for the Utah CHESS communities are given in Tables 2.1.A.3 through 2.1.A.7. Cumulative frequency distributions of daily sulfur dioxide concentrations in 1971 exhibited the expected differences across communities (Figure 2.1.6). In Magna and Kearns, which are near the smelter, concentrations were highly dependent on wind direction, as reflected in the steep slopes in Figure 2.1.6. Smelter emissions would usually be quite diluted and probably overshadowed by local emission sources in Salt Lake City and Ogden, resulting in lower peak exposures and in lower peak sulfur dioxide concentrations in these cities. The gradual slopes in Figure 2.1.6 for Salt Lake City and Ogden reflect internal sources rather than smelter emissions.

Annual average concentrations in all four communities are below the National Annual Primary Standard (80 $\mu g/m^3$). Magna, which also has the highest annual average sulfur dioxide concentration (61.8 $\mu g/m^3$), exceeded the National Daily Air Quality Standard (365 $\mu g/m^3$) on 1 percent of the days.

The total suspended particulate frequency distribution is shown in Figure 2.1.7. Magna, Ogden, and Salt Lake City rarely exceeded the daily standard (260 $\mu g/m^3$); Kearns did not approach the daily standard. The pattern of community exposure to particulates differed from the pattern of exposure to sulfur dioxide. Particulate concentrations were highest in Salt Lake City and Ogden, while sulfur dioxide concentrations were highest in Magna and Kearns.

Gaseous sulfur dioxide reacts with atmospheric components to form suspended sulfates. A cumulative frequency distribution of daily suspended sulfate concentrations is shown in Figure 2.1.8. The gradient of daily community exposure from highest to lowest - Magna, Kearns, Salt Lake City, and Ogden - is the same as that shown for sulfur dioxide in Figure 2.1.6.

### Monthly Concentrations

Monthly arithmetic mean sulfur dioxide concentrations (Figure 2.1.9) showed, as expected, that Magna had the highest sulfur dioxide concentration, with Kearns, Salt Lake City, and Ogden following in that order. Due to seasonal differences in inversion frequency, winter months generally had higher sulfur dioxide concentrations than summer months. A strike at the smelter during the month of July was reflected in an extremely low level of sulfur dioxide in Magna and Kearns.

Monthly geometric mean suspended sulfate concentrations (Figure 2.1.10) revealed a community gradient similar to that of sulfur dioxide. Again summer had lower levels than winter. The effect of the strike in July was again manifested in Magna and Kearns.

Maximum daily sulfur dioxide concentrations for each month are shown in Figure 2.1.11. Comparison of Figure 2.1.9 and 2.1.11 reveals that although Salt Lake City, Ogden, and Kearns had similar monthly arithmetic mean sulfur dioxide concentrations, Kearns had considerably higher peak values. Peak values for Kearns, however, were considerably lower than those for Magna.

Salt Lake City had the highest monthly total suspended particulate concentration (Figure 2.1.12). Ogden, Magna, and Kearns followed in that order. The higher values in Ogden and Salt Lake City can possibly be explained by the location of monitoring sites in areas of fairly heavy automotive traffic. Summer months again showed lower levels of total suspended particulate.

Salt Lake City had the highest monthly nitrogen dioxide concentration (Figure 2.1.13), with Ogden following close behind. Kearns and Magna had similar but considerably lower concentrations. The higher concentrations in Salt Lake City and Ogden can possibly be explained by site locations in areas of heavy motor vehicle traffic. Nitrogen dioxide concentrations fell markedly during the summer months.

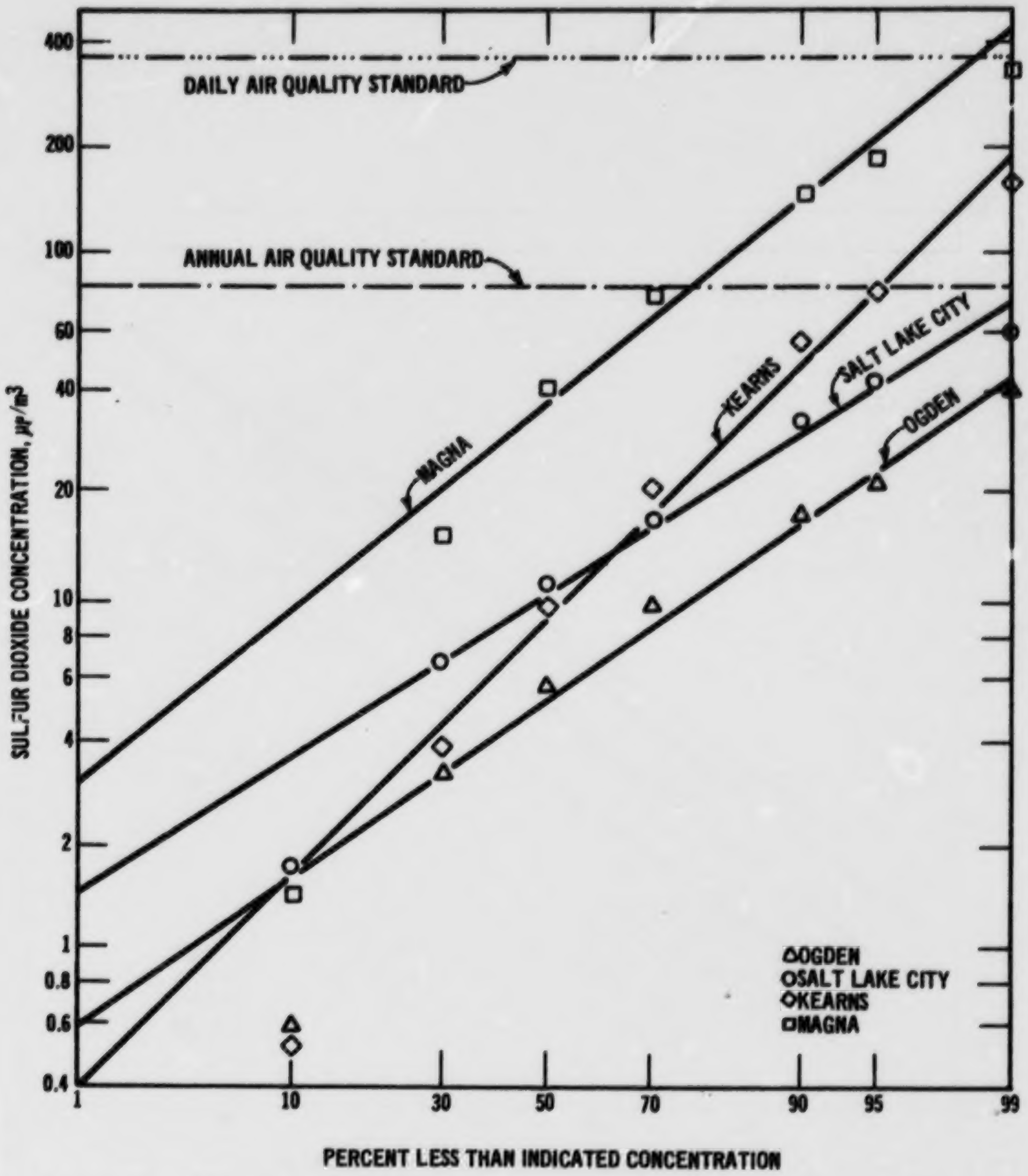

Figure 2.1.6 Cumulative frequency of sulfur dioxide concentrations for Utah CHESS communities.

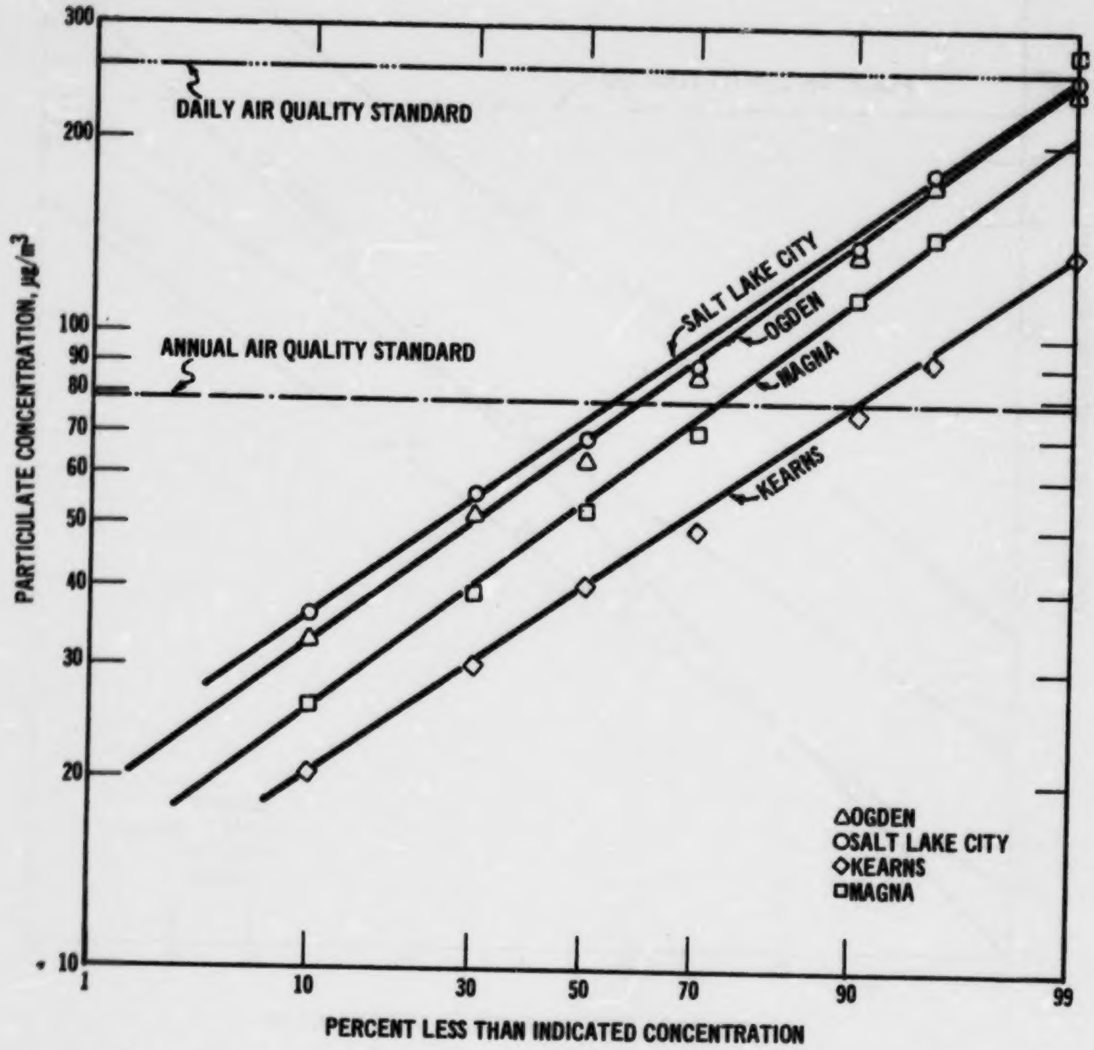

Figure 2.1.7. Cumulative frequency of total suspended particulate concentrations for Utah CHESS communities.

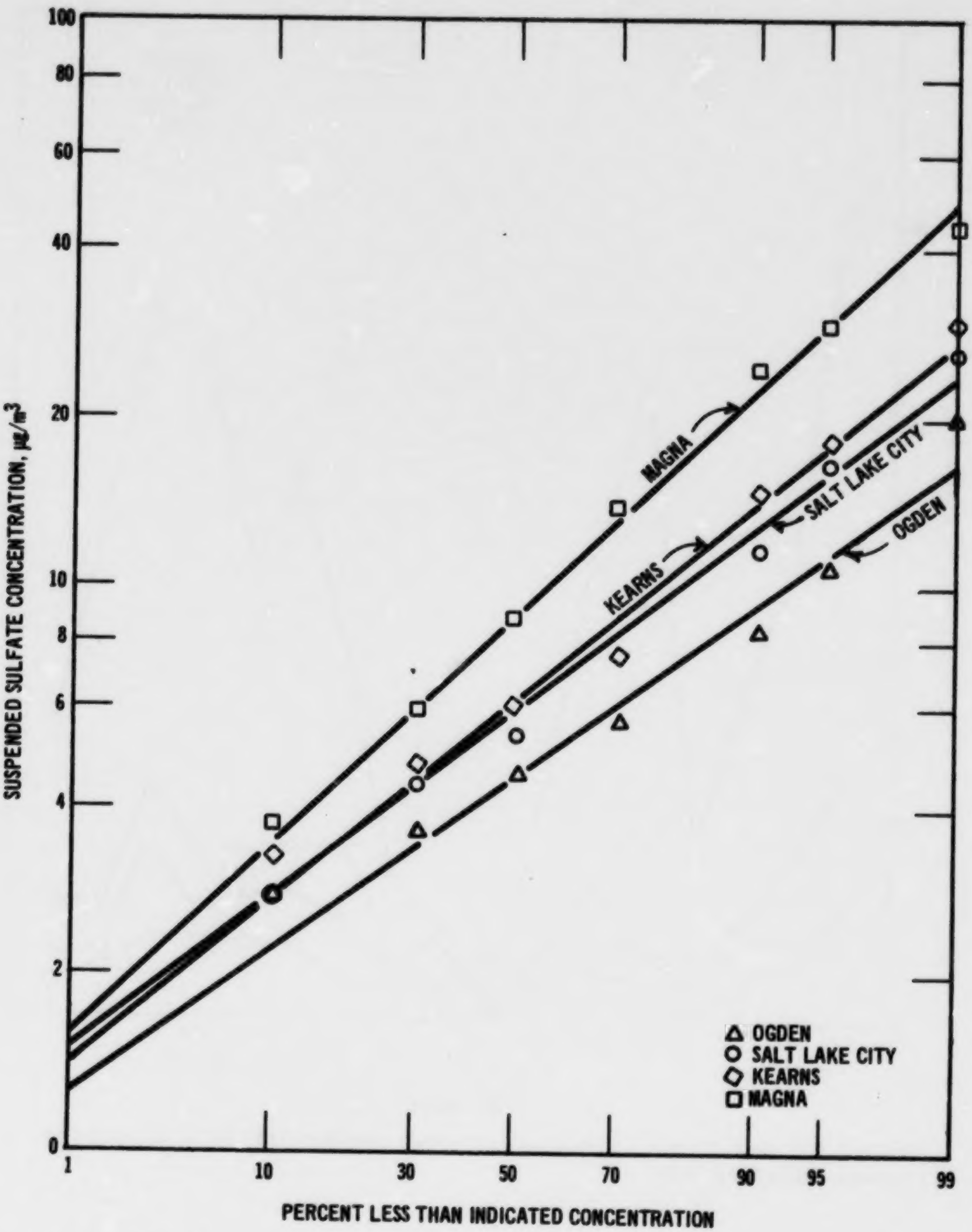

Figure 2.1.8. Cumulative frequency of suspended sulfate concentrations for Utah CHESS communities.

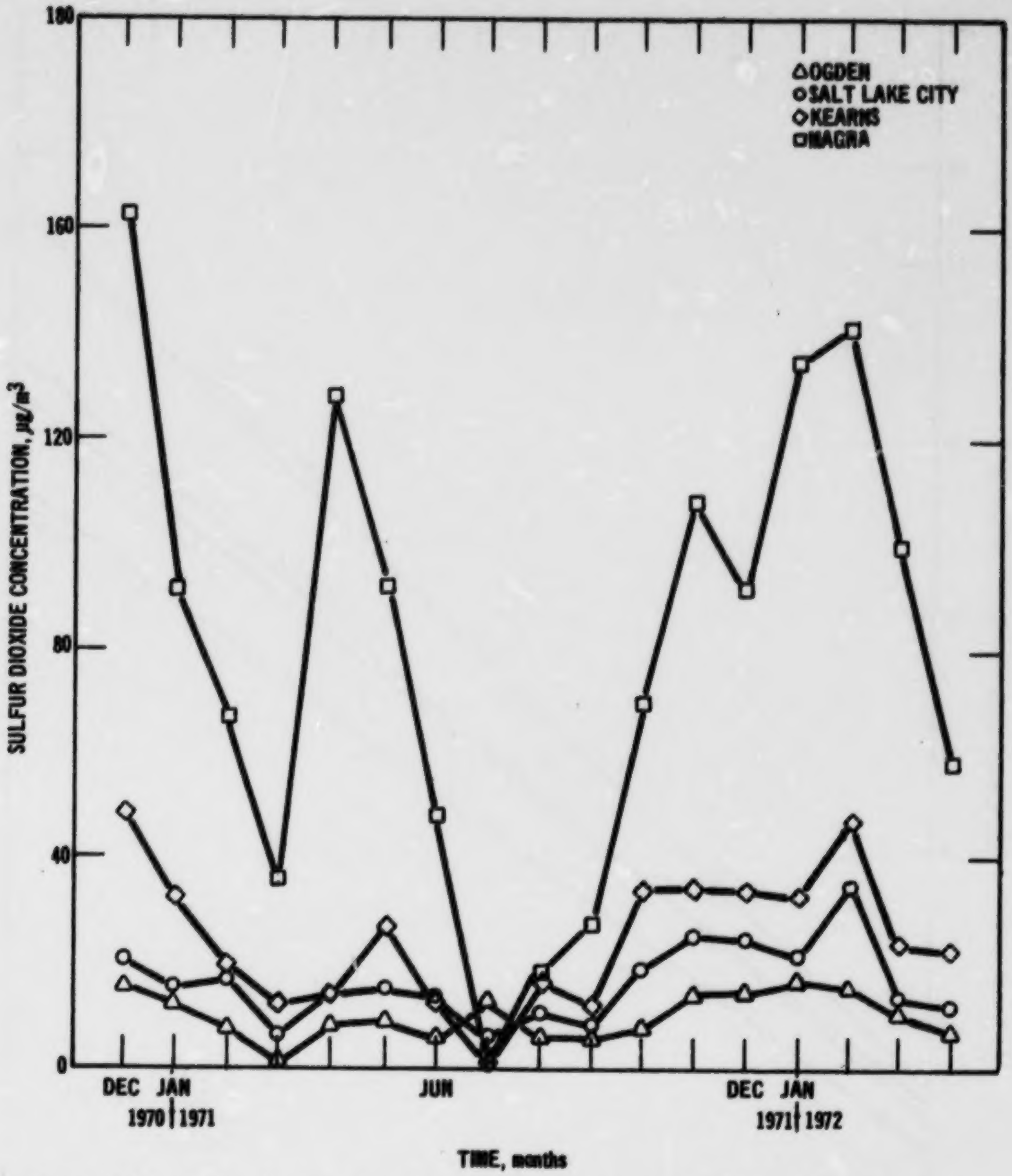

Figure 2.1.9. Sulfur dioxide concentrations versus time for Utah CHESS communities. December 1970-April 1972.

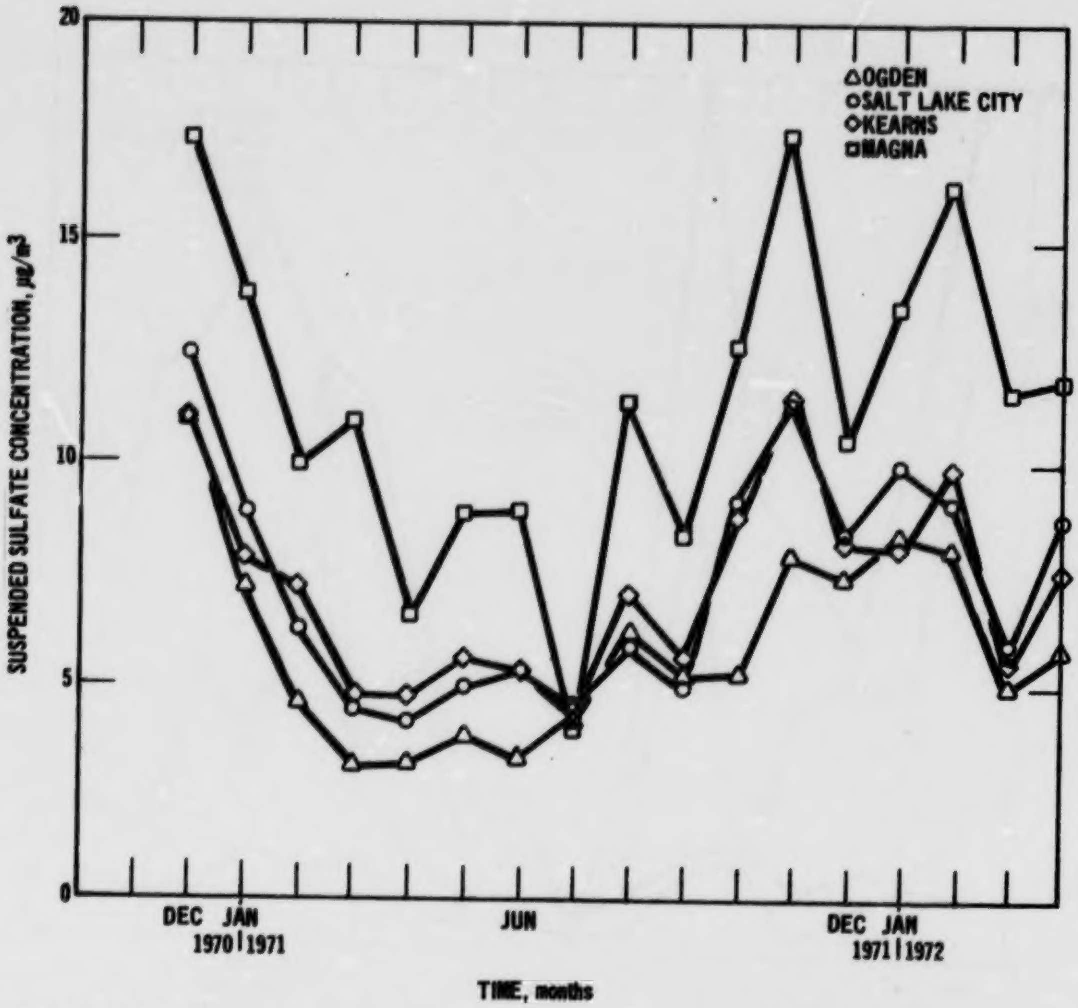

Figure 2.1.10. Suspended sulfate concentrations versus time for Utah CHESS communities, December 1970 - April 1972.

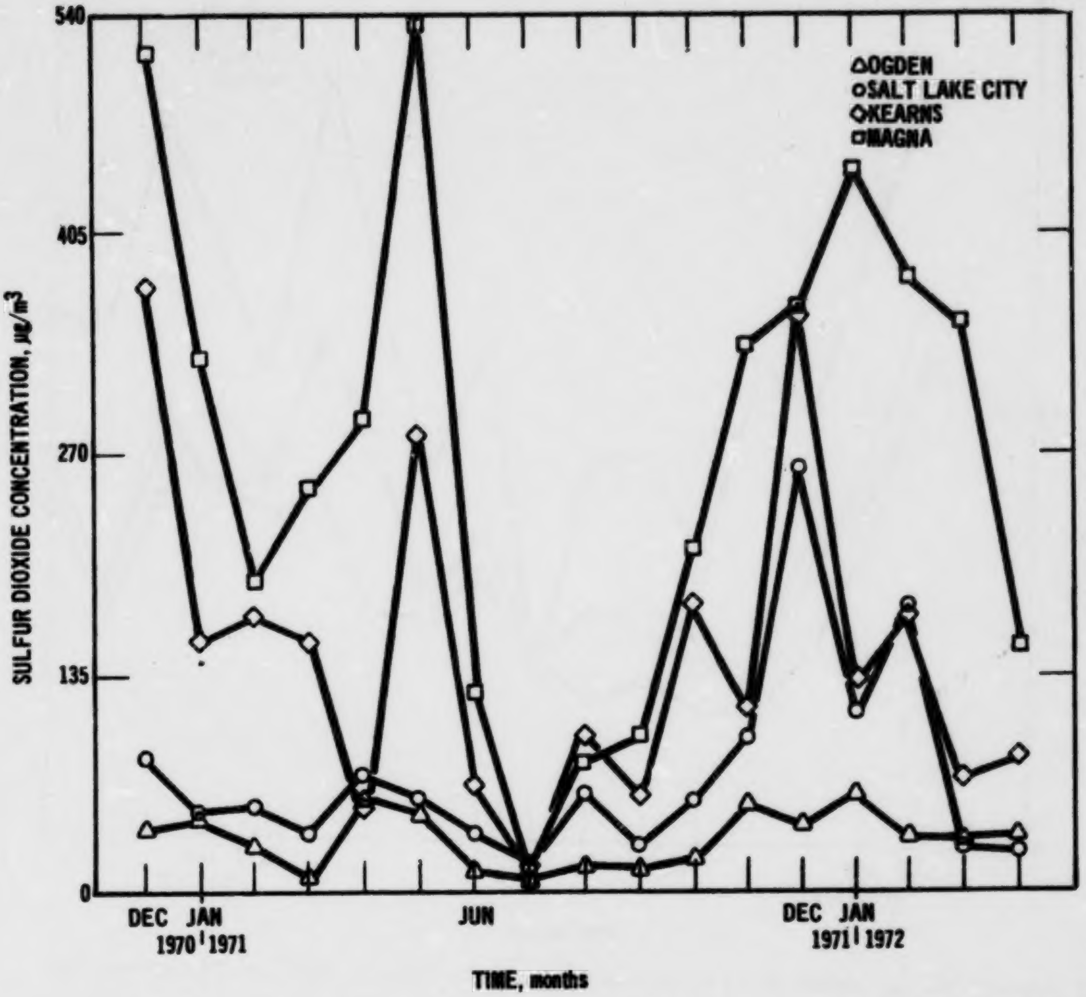

Figure 2.1.11. Maximum 24-hour sulfur dioxide concentrations versus time for Utah CHESS communities, December 1970 - April 1972.

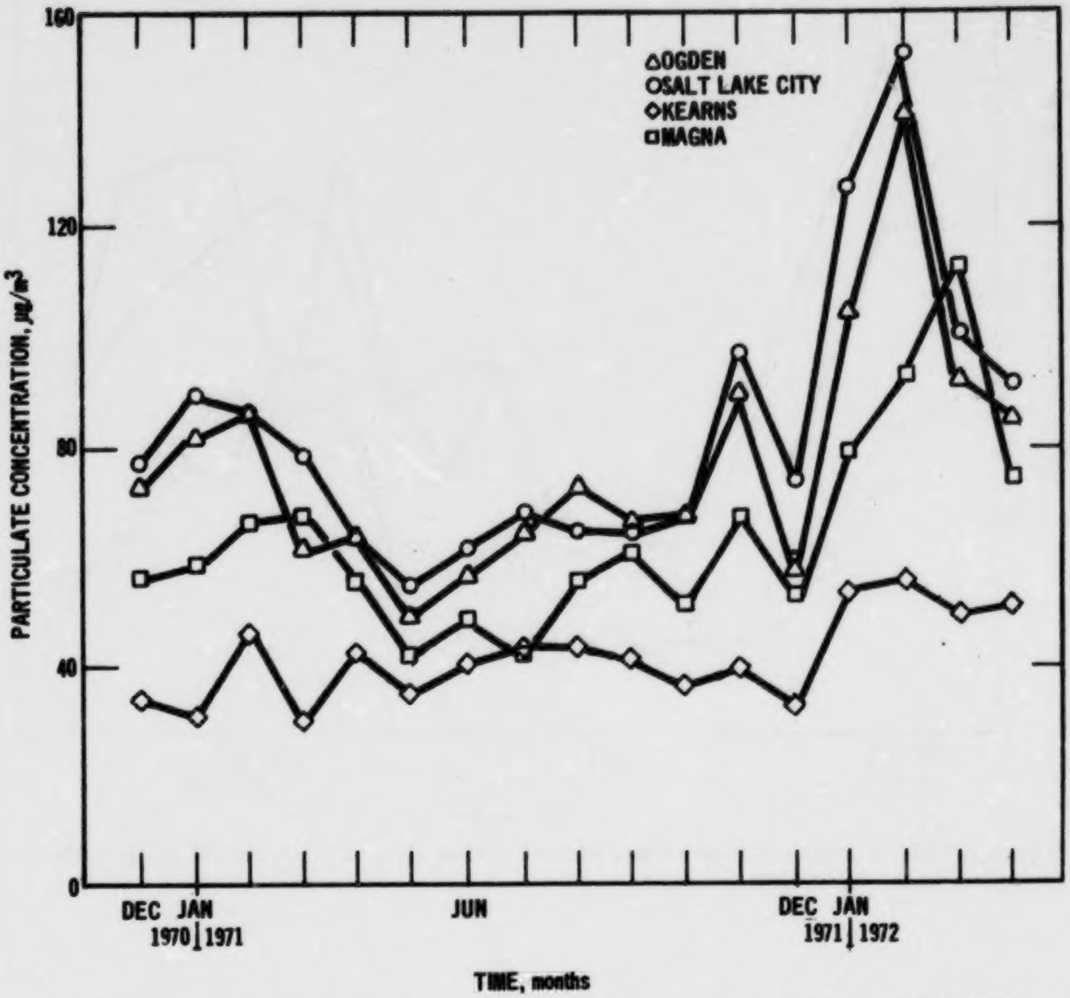

Figure 2.1.12. Total suspended particulate concentrations versus time for Utah CHESS communities, December 1970 - April 1972.

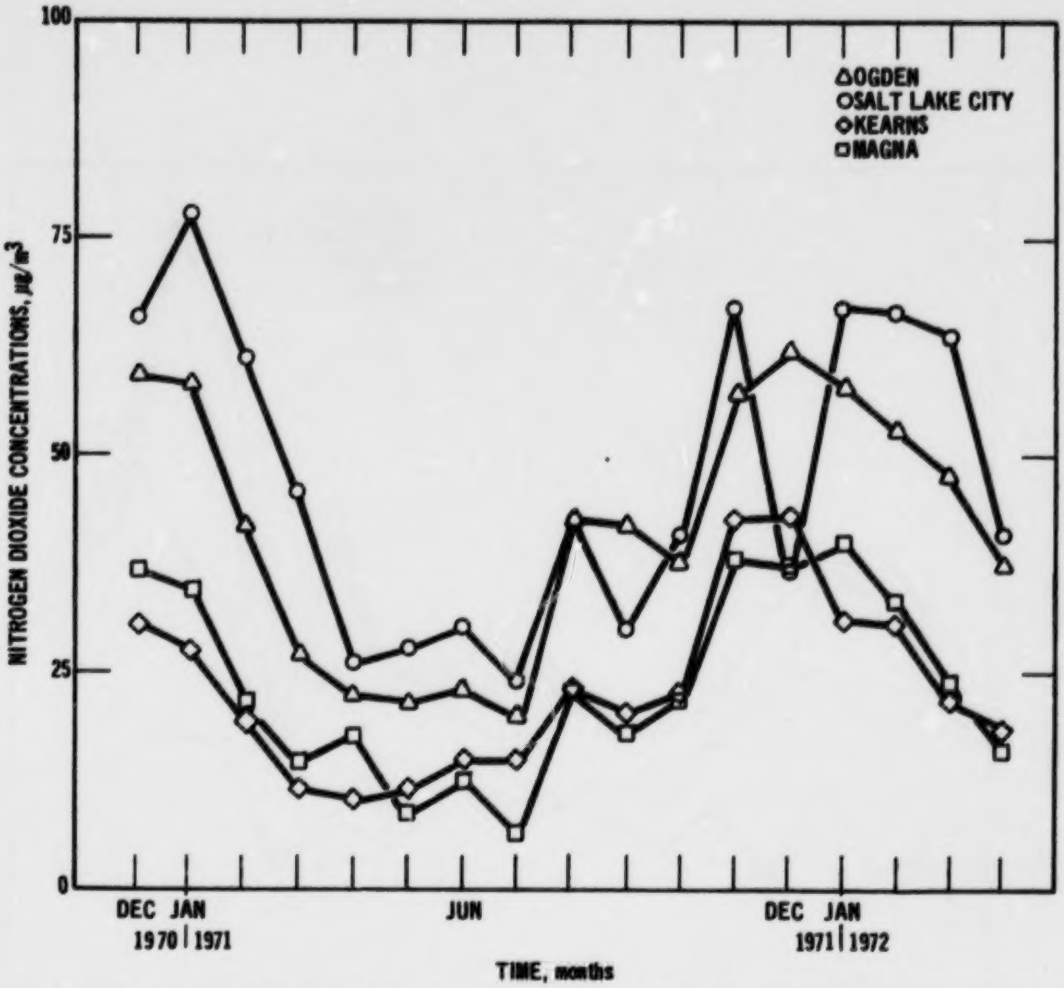

Figure 2.1.13. Nitrogen dioxide concentrations versus time for Utah CHESS communities, December 1970 - April 1972.

The gradient and seasonal patterns for suspended nitrates (Figure 2.1.14) were the same as those for nitrogen dioxide. Lowest suspended nitrate concentrations, however, were recorded in February and March.

Monthly arithmetic mean COH levels (Figure 2.1.15) followed the same gradient as total suspended particulates, with Salt Lake City having the highest level. COH levels were considerably lower during summer months.

No consistent area or seasonal patterns for monthly dustfall or for cadmium, lead, and zinc in dustfall (Tables 2.1.A.8 through 2.1.A.11) could be observed.

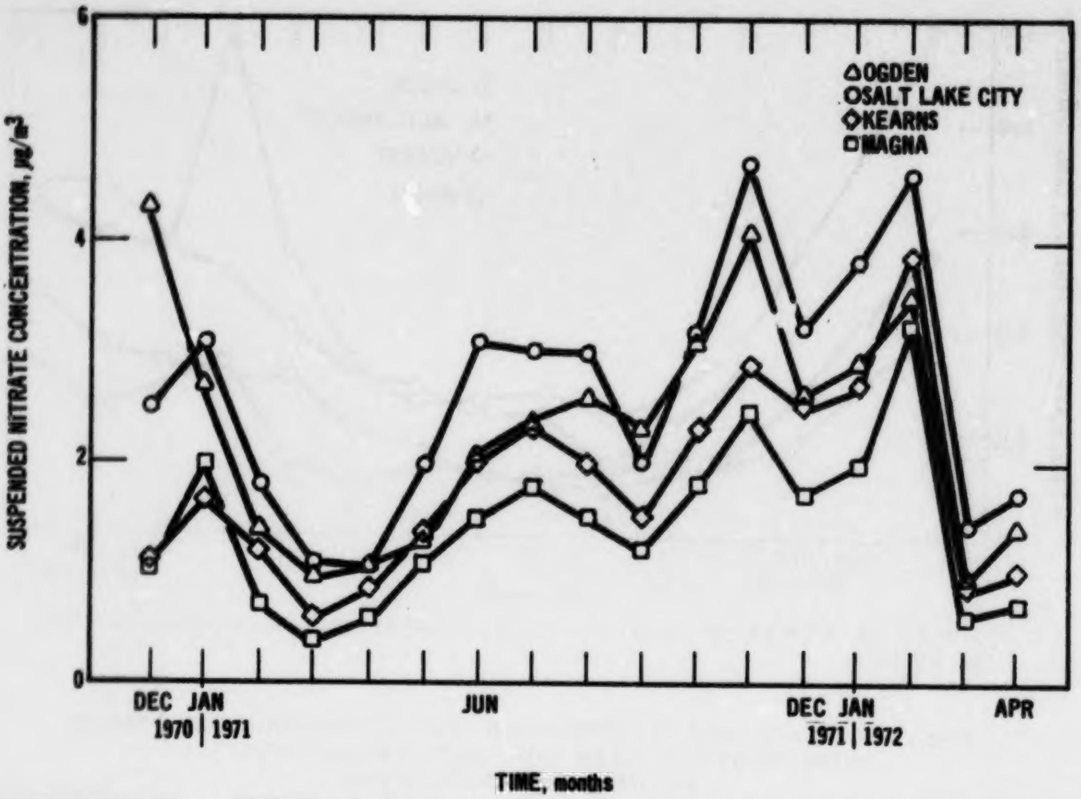

Figure 2.1.14. Suspended nitrate concentrations versus time for Utah CHESS communities, December 1970 - April 1972.

## Interrelationships among Pollutants

Prior studies have been conducted to determine the relationship between various pollutant concentrations. In order to systematically examine these relationships for the Utah communities, a set of correlation matrices was calculated (Tables 2.1.A.12 and 2.1.A.13). These matrices present the correlation coefficients between a specific pollutant at a given site and another pollutant and site.

To determine the relationship between suspended sulfates and sulfur dioxide, regression lines and correlation coefficients were calculated for daily averages of suspended sulfate against corresponding values of sulfur dioxide. The calculations for the Utah CHESS communities (Table 2.1.4) demonstrate reasonably high correlation coefficients between suspended sulfates and sulfur dioxide in Magna, Kearns, and Ogden (0.67, 0.58, and 0.46, respectively). A low correlation (0.31) was found in Salt Lake City, which had the largest number of small sulfur dioxide emission sources.

Similar calculations were made for suspended sulfates and total suspended particulates (Table 2.1.4). Correlations were quite similar for all four communities (0.50, 0.53, 0.57, and 0.48 for Magna, Kearns, Salt Lake City, and Ogden, respectively). The reasons for the observed relationships are not clear. One possible explanation is that a large point emission source of sulfur dioxide also generates a large amount of suspended sulfate, resulting in a high correlation between the two. In an area like Salt Lake City, where there are multiple urban sources of sulfur dioxide, suspended sulfate concentrations may become a complex function of sulfur oxide and suspended particulate concentrations.

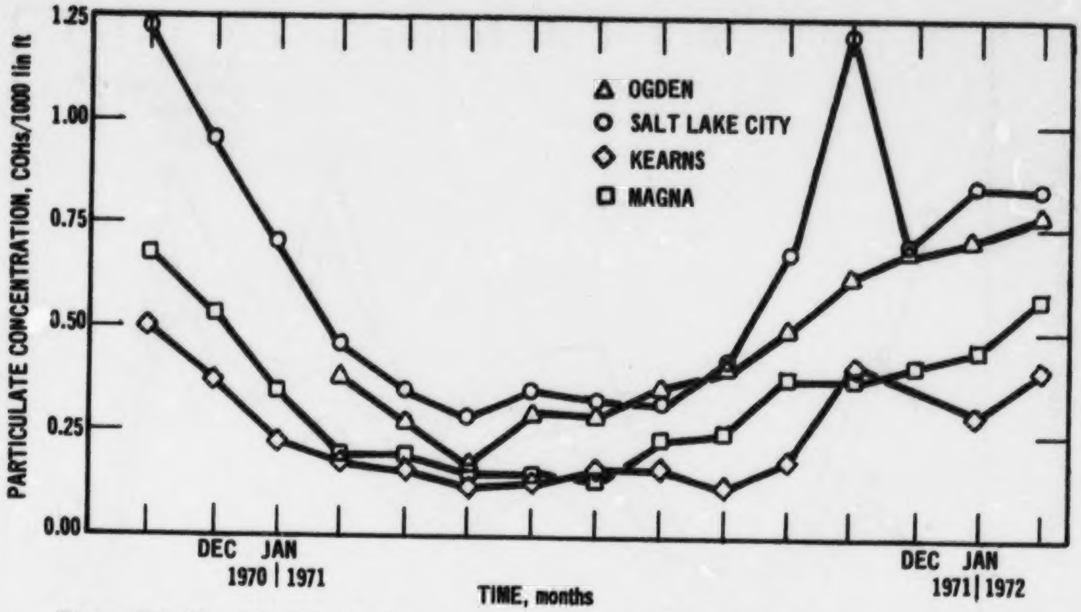

Figure 2.1.15. COH levels versus time for Utah CHESS communities, December 1970 - February 1972.

Table 2.1.4. RELATIONSHIP OF SUSPENDED SULFATES TO SULFUR DIOXIDE AND OF SUSPENDED SULFATES TO TOTAL SUSPENDED PARTICULATES FOR UTAH CHESS COMMUNITIES

| Relationship | Magna | Kearns | Salt Lake City | Ogden |
|---|---|---|---|---|
| Suspended sulfate versus sulfur dioxide: | | | | |
| Intercept | 6.66 | 5.41 | 5.86 | 3.86 |
| Slope | 0.09 | 0.10 | 0.09 | 0.20 |
| Correlation coefficient | 0.67 | 0.58 | 0.31 | 0.46 |
| Suspended sulfate versus total suspended particulate: | | | | |
| Intercept | 6.39 | 1.84 | 1.93 | 2.59 |
| Slope | 0.09 | 0.13 | 0.065 | 0.038 |
| Correlation coefficient | 0.50 | 0.53 | 0.57 | 0.48 |

## Long-term Exposure Trends

Past exposures to sulfur dioxide and suspended sulfates were calculated from smelter emission estimates dating back to 1940 (Table 2.1.A.14). The rate of measured annual sulfur dioxide concentration at Magna to smelter emission in 1971 (61.8/193) was multiplied by the smelter emissions for each year prior to 1971 to obtain estimates of annual sulfur dioxide concentrations from 1940 to 1970. Similar calculations were made from the 1971 observed sulfur dioxide concentration in Kearns. Sulfur dioxide values obtained by the State of Utah in 1970 and 1971 were averaged, and the ratio of this value to the average smelter emission in 1970-1971 was used to obtain another estimate of sulfur dioxide exposure from 1940-1969. The ratio of sulfur dioxide concentration for Kearns for 1971 obtained by the State of Utah to the 1971 smelter emission allowed another estimate of past exposure for Kearns to be made. The conductivity method used by the State of Utah yields higher sulfur dioxide results because salts and other electrolytes affect instrument response. Both sets of estimates were compared with estimates obtained by employing a mathematical diffusion model developed by the National Environmental Research Center's Division of Meteorology. The diffusion model made use of smelter emission data, wind roses, and topographical characteristics of the area to compute contours of sulfur dioxide concentrations in the vicinity of Magna.

Although production of copper has increased from 1940 to 1971, emissions have decreased from 970 tons/day in 1940 to 193 tons/day in 1971 (Table 2.1.A.14). Two factors account for this reduction. First, the number of sulfuric acid plants utilizing sulfur recovered from emissions have increased from one in 1940 to seven in 1971. Second, air pollution control devices in the form of baghouses, scrubbers, cyclones, and mist eliminators have been installed.

Theoretical sulfur dioxide contours around the smelter source (Figure 2.1.16) were determined by applying a mathematical diffusion model, the Air Quality Display Model (AQDM).[15] As suspected, the up-valley and down-valley wind pattern in the region resulted in maximum downwind concentrations to the north-northwest. Reversal of wind direction to up-slope in the afternoon caused high concentrations in the south to southeast. These wind patterns resulted in an elongated pattern of sulfur dioxide contours. Fortunately, no populated areas are located near the centers of concentrations since Great Salt Lake lies to the northwest and the Oquirrh Mountains to the south-southeast. The pattern displayed in Figure 2.1.16 shows Magna located on the fringe of the contours with a theoretical concentration of 41

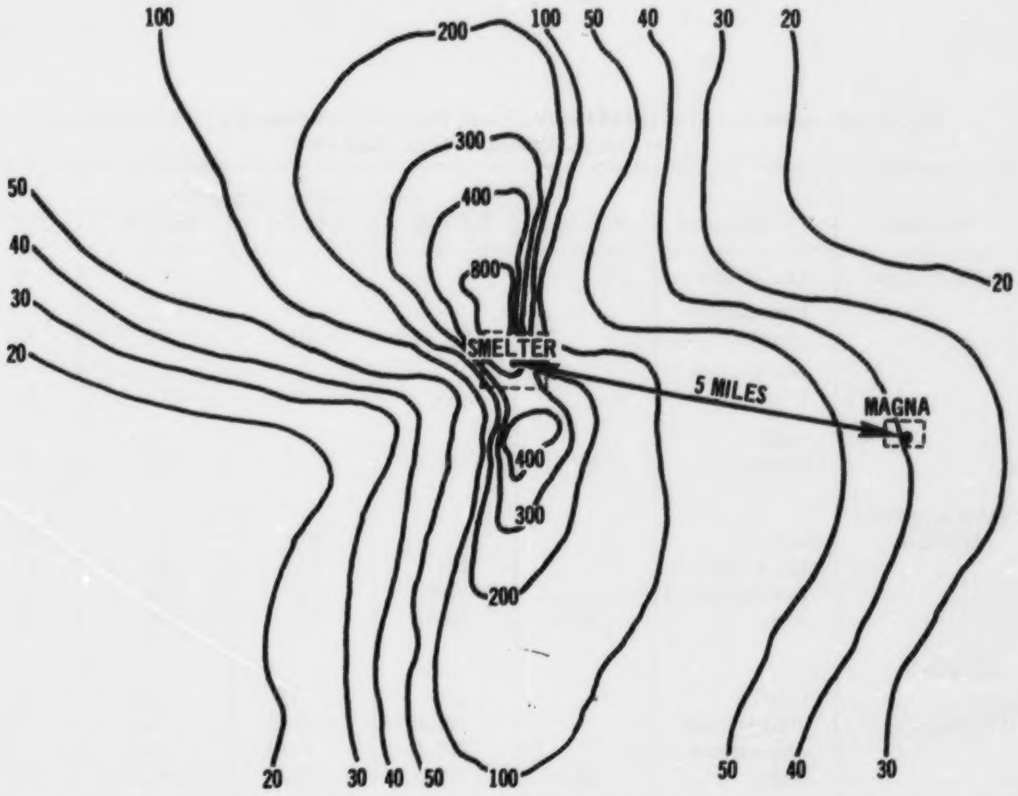

Figure 2.1.16. Sulfur dioxide concentration (μg/m$^3$) contours based on mathematical diffusion model for 1971 smelter emission rate of 193 tons/day.

μg/m³ for the 1971 emission rate of 193 tons/day. Measurements of sulfur dioxide by the CHESS program revealed a concentration of 62 μg/m³ for the same year. The excess of observed over predicted values may be accounted for by emissions from sulfuric acid plants near Magna. No emissions data were available from these plants, and thus they could not be factored into the diffusion model. The diffusion model was useful in demonstrating that annual exposure estimates obtained from the ratio of 1971 observed air quality to 1971 emissions were not unreasonably high or low.

Annual average total suspended particulate data for Salt Lake City from the National Air Surveillance Network are available back to 1953, whereas corresponding sulfur dioxide data are available only back to 1962. In order to obtain another estimate of the sulfur dioxide concentrations for the years 1953 to 1961, an average ratio of sulfur dioxide to total suspended particulate for the available Salt Lake data was used.

To estimate total suspended particulate concentrations in Magna and Kearns, the ratios of Magna's and Kearn's 1971 observed concentrations (66.2 and 44.5 μg/m³) to the 1971 production of copper for the Garfield smelter (260,000 tons) were calculated. These ratios were multiplied by the production figure for each year back to 1940 to give annual estimates of particulate exposure. Annual production figures are given in Table 2.1.A.15.[16]

Actual and estimated air quality data for the Utah CHESS communities are compiled in Table 2.1.A.16. Table 2.1.5 summarizes the estimates of long-term pollutant exposure given in Table 2.1.A.16 and identifies the communities according to the exposure gradient established for sulfur dioxide and suspended sulfates. Clearly, pollution gradients of sulfur oxide exposure are evident between each of the four locations and between the three decades in time.

The graphs of sulfur dioxide and suspended sulfate concentrations shown in Figures 2.1.17 and 2.1.18

**Table 2.1.5. ANNUAL ARITHMETIC AVERAGE POLLUTANT CONCENTRATIONS FOR UTAH CHESS COMMUNITIES, 1940-1971**

| Pollutant | Community | Concentration,[a] μg/m³ 1940-49 | 1950-59 | 1960-69 | 1965-70 | 1971 |
|---|---|---|---|---|---|---|
| Sulfur dioxide | Low (Ogden) | – | – | – | – | 8 |
| | Intermediate I (Salt Lake City) | – | – | (20) | 17 | 15 |
| | Intermediate II (Kearns)[b] | – | (50) | (33) | (33) | 22 |
| | High (Magna) | (234) | (142) | (95) | (93) | 62 |
| Total suspended particulate | Low | – | – | 100 | 97 | 78 |
| | Intermediate I | – | 151 | 112 | 89 | 81 |
| | Intermediate II | – | (40) | (38) | (43) | 45 |
| | High | (63) | (60) | (57) | (64) | 66 |
| Suspended sulfates | Low | – | – | 4.8 | 4.0 | 5.6 |
| | Intermediate I | – | (11.4) | (7.5) | 5.7 | 7.3 |
| | Intermediate II | – | (10.4) | (8.8) | (8.7) | 7.8 |
| | High | (27.7) | (19.5) | (15.3) | (15.0) | 12.4 |

[a] Parentheses indicate estimated values.

[b] Kearns, the Intermediate II community, was established in 1953.

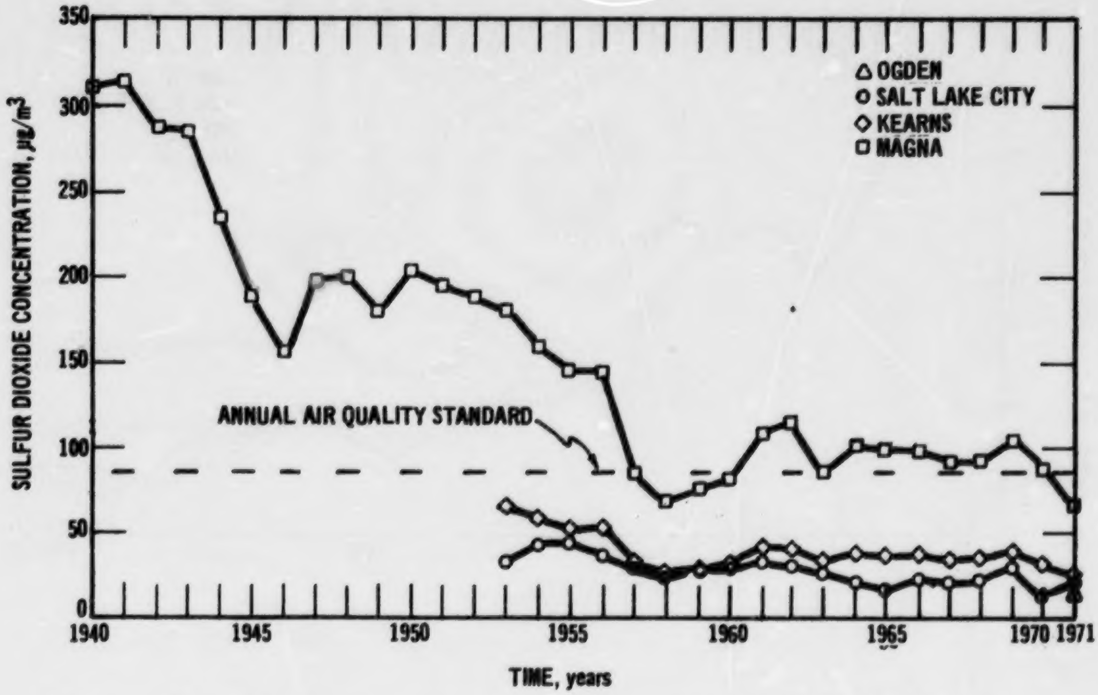

Figure 2.1.17. Sulfur dioxide concentrations versus time for Utah CHESS communities, 1940-1971.

indicate that previous sulfur dioxide concentrations have been considerably in excess of the present National Primary Air Quality Standard. Figure 2.1.19 indicates that total suspended particulate values in Salt Lake City and Ogden have been considerably above the present National Primary Air Quality Standard. The historical values of total suspended particulate for Magna and Kearns have been relatively low.

## SUMMARY

The position of the CHESS communities in the Salt Lake Basin relative to the major copper smelter and the local meteorological pattern provided an area gradient of exposure to sulfur oxides. The exposures decreased with distance from the point source.

The city of Magna, Utah, located in close proximity to the smelter, had high exposure to sulfur oxides. Although the prevailing winds transport source emissions away from Magna most of the time, concentrations were estimated to exceed the National Primary Air Quality Standard for sulfur dioxide (80 µg/m³) during more than 75 percent of the past 32 years on an annual basis. Increased utilization of sulfur oxides by the copper smelter for the manufacture of sulfuric acid and installation of control devices resulted in a downward trend in emissions and ambient air concentrations of sulfur dioxide at Magna.

Kearns, Salt Lake City, and Ogden, Utah, in that order, yielded a descending exposure gradient to sulfur oxides. Magna and Kearns experienced a greater frequency of short-term high concentrations of sulfur dioxide since they were closer to the smelter source. Correlations between sulfur dioxide and suspended sulfates were higher in Magna and Kearns than in Salt Lake City and Ogden.

Air quality measurements made in the Utah CHESS residential areas in 1971 did not exceed the National Annual Ambient Air Quality Standards. CHESS air monitoring sites were purposely located in close proximity to the population under study and

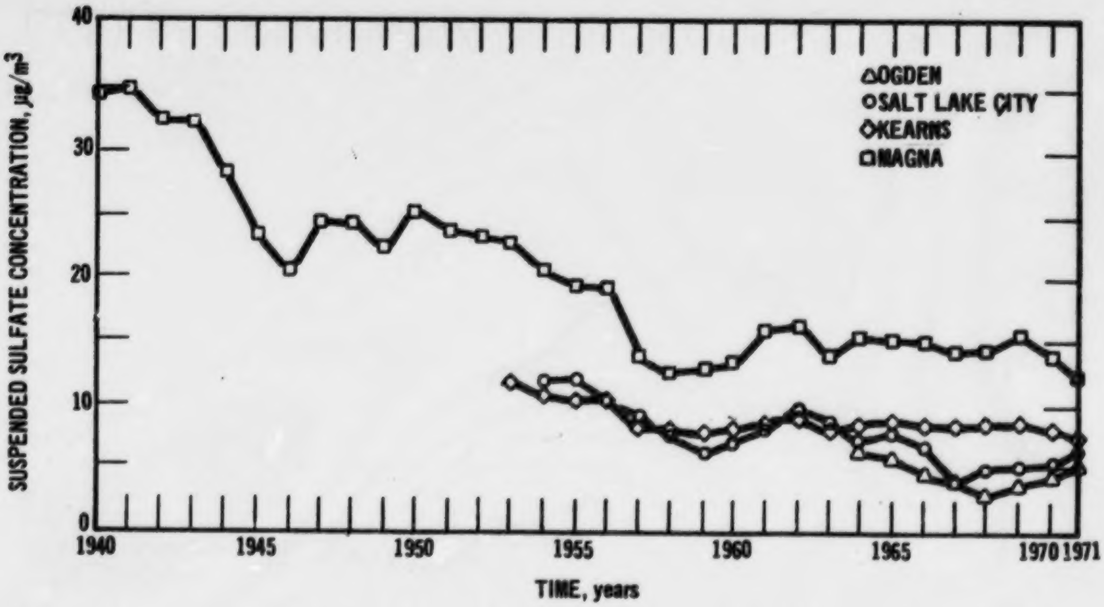

Figure 2.1.18. Suspended sulfate concentrations versus time for Utah CHESS communities, 1940-1971.

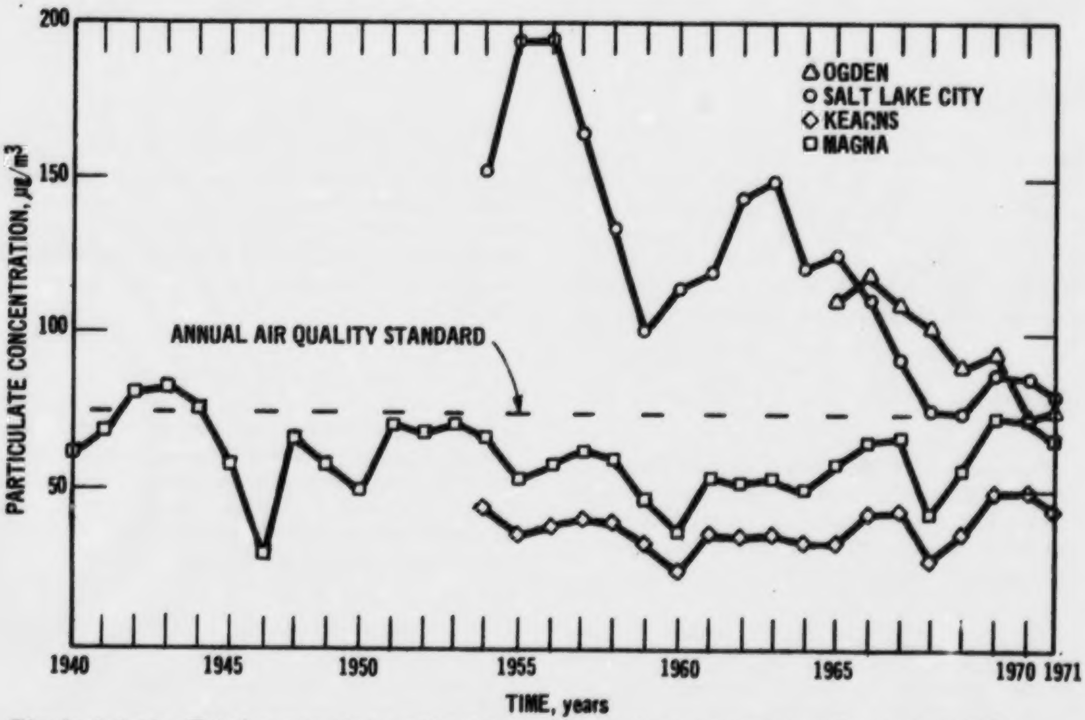

Figure 2.1.19. Total suspended particulate concentrations versus time for Utah CHESS communities, 1940-1971.

therefore represented average exposure of the neighborhoods.

Estimates of past exposures to sulfur dioxide dating to 1940 were derived on the basis of annual smelter emissions. These estimates indicated that past exposures were considerably higher (about 120 percent higher in the 1950's) than current levels. Although past exposure estimates are admittedly crude, they do offer some guidance in determining past human exposure to air pollutants.

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## APPENDIX

### Table 2.1.A.1. TOTAL SUSPENDED PARTICULATE CONCENTRATION FROM UTAH STATE DIVISION OF HEALTH, 1963-1971

| Community | Year | Particulate concentration,[a] μg/m$^3$ |
|---|---|---|
| Ogden | 1965 | 102 |
| | 1966 | 84 |
| | 1967 | 92 |
| | 1968 | 78 |
| | 1969 | 84 |
| | 1970 | 69 |
| | 1971 | 78 |
| Salt Lake City | 1963 | 121 |
| | 1964 | 112 |
| | 1965 | 104 |
| | 1966 | 95 |
| | 1967 | 79 |
| | 1968 | 85 |
| | 1969 | 90 |
| | 1970 | 84 |
| | 1971 | 94 |
| Kearns | 1971 | 51 |
| Magna | 1968 | 70 |
| | 1969 | 65 |
| | 1970 | 70 |
| | 1971 | 71 |

[a] Concentrations are annual geometric mean values for daily 24-hour averages. Data since 1970 have been corrected for annual average ambient barometric pressure and daily average ambient temperature at the sampling site and apply at standard temperature and pressure.

### Table 2.1.A.2. SULFUR OXIDE CONCENTRATION FROM UTAH STATE DIVISION OF HEALTH, 1965-1971

| Community | Year | Sulfur oxide concentration,[a] μg/m$^3$ |
|---|---|---|
| Ogden | 1965 | 5.2 |
| | 1966 | 2.6 |
| | 1967 | 5.2 |
| | 1968 | 2.6 |
| | 1969 | 2.6 |
| | 1970 | 2.6 |
| | 1971 | 10.5 |
| Salt Lake City | 1965 | 49.8 |
| | 1966 | 41.9 |
| | 1967 | 5.2 |
| | 1968 | 7.9 |
| | 1969 | 10.5 |
| | 1970 | 15.7 |
| | 1971 | 21.0 |
| Kearns | 1971 | 34.1 |
| Magna | 1970 | 120.5 |
| | 1971 | 107.4 |

[a] Concentrations are annual arithmetic mean values. Averages for 1965-1969 are from the National Aerometric Data Bank printouts based on Utah State Division of Health Data submitted to the Mitre Corporation in 1969. Data since 1970 have been corrected for average ambient barometric pressure at the sampling site.

## Table 2.1.A.3. DAILY SULFUR DIOXIDE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - MARCH 1972

| Community | Period of observation | Observations | Concentration, $\mu g/m^3$ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standard,[b] % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Ogden | 11/70-12/70 | 13 | 2.9 | 36.7 | 15.8 | 12.5 | 0.74 | 6.0 | 8.4 | 12.7 | 18.9 | 32.8 | 35.4 | 36.4 | 0.0 | 0.0 |
|  | 1/71- 3/71 | 81 | 0.0 | 43.3 | 7.4 | 5.1 | 1.10 | 0.0 | 1.8 | 4.5 | 8.7 | 18.4 | 24.5 | 36.2 | 0.0 | 0.0 |
|  | 4/71- 6/71 | 87 | 0.0 | 59.1 | 7.8 | 6.4 | 0.83 | 0.0 | 3.6 | 5.8 | 8.6 | 16.6 | 18.5 | 49.3 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 83 | 0.0 | 12.6 | 4.6 | 4.0 | 0.76 | 0.4 | 2.4 | 4.1 | 6.1 | 9.2 | 10.6 | 11.8 | 0.0 | 0.0 |
|  | 10/71-12/71 | 87 | 1.4 | 50.5 | 12.0 | 9.2 | 0.77 | 3.2 | 6.5 | 9.9 | 15.2 | 21.5 | 28.9 | 41.0 | 0.0 | 0.0 |
|  | 1/71-12/71 | 338 | 0.0 | 59.1 | 8.0 | 6.0 | 0.92 | 0.6 | 3.0 | 5.7 | 9.4 | 17.5 | 21.5 | 41.9 | 0.0 | 0.0 |
|  | 1/72- 3/72 | 85 | 1.0 | 58.2 | 14.2 | 10.8 | 0.80 | 4.1 | 8.2 | 11.8 | 15.6 | 26.0 | 32.5 | 48.2 | 0.0 | 0.0 |
| Salt Lake | 11/70-12/70 | 34 | 0.0 | 79.4 | 17.4 | 10.1 | 1.25 | 0.9 | 5.6 | 9.9 | 22.5 | 40.9 | 56.5 | 76.6 | 0.0 | 0.0 |
|  | 1/71- 3/71 | 68 | 0.0 | 51.6 | 13.3 | 9.6 | 1.06 | 0.0 | 4.0 | 9.5 | 16.5 | 34.2 | 39.1 | 49.0 | 0.0 | 0.0 |
|  | 4/71- 6/71 | 88 | 0.0 | 70.7 | 15.2 | 12.0 | 0.79 | 3.0 | 7.5 | 12.8 | 17.9 | 30.8 | 33.4 | 58.7 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 62 | 0.0 | 58.7 | 8.9 | 6.3 | 1.04 | 0.7 | 4.8 | 7.4 | 9.9 | 16.4 | 19.1 | 40.5 | 0.0 | 0.0 |
|  | 10/71-12/71 | 70 | 0.3 | 259.3 | 23.0 | 13.8 | 1.04 | 5.1 | 10.1 | 13.8 | 20.4 | 49.4 | 59.7 | 143.7 | 2.9 | 0.0 |
|  | 1/71-12/71 | 288 | 0.0 | 259.3 | 15.3 | 10.3 | 1.01 | 1.6 | 6.6 | 10.6 | 16.6 | 32.4 | 43.2 | 66.0 | 0.7 | 0.0 |
|  | 1/72- 3/72 | 84 | 0.0 | 175.5 | 23.7 | 15.7 | 0.94 | 4.7 | 9.9 | 14.9 | 20.4 | 50.3 | 66.6 | 122.8 | 4.8 | 0.0 |
| Kearns | 11/70-12/70 | 37 | 0.0 | 367.3 | 40.3 | 18.5 | 1.71 | 0.0 | 1.8 | 13.2 | 42.2 | 101.8 | 118.2 | 292.0 | 16.2 | 2.7 |
|  | 1/71- 3/71 | 75 | 0.0 | 166.9 | 21.5 | 10.9 | 1.28 | 0.2 | 3.8 | 10.0 | 17.3 | 52.0 | 115.5 | 156.4 | 6.7 | 0.0 |
|  | 4/71- 6/71 | 86 | 0.0 | 277.2 | 18.8 | 10.1 | 1.25 | 0.6 | 4.2 | 9.5 | 17.5 | 47.3 | 60.7 | 107.5 | 1.2 | 0.0 |
|  | 7/71- 9/71 | 80 | 0.0 | 94.6 | 10.9 | 5.9 | 1.42 | 0.0 | 1.2 | 4.4 | 13.5 | 22.1 | 43.1 | 69.8 | 1.2 | 0.0 |
|  | 10/71-12/71 | 87 | 0.0 | 357.5 | 34.7 | 16.1 | 1.44 | 1.3 | 7.9 | 18.9 | 39.4 | 76.3 | 101.6 | 199.9 | 9.2 | 0.0 |
|  | 1/71-12/71 | 328 | 0.0 | 357.5 | 21.7 | 10.4 | 1.39 | 0.5 | 3.6 | 9.7 | 20.1 | 56.1 | 76.5 | 165.4 | 4.6 | 0.0 |
|  | 1/72- 3/72 | 89 | 0.0 | 167.4 | 34.7 | 22.6 | 1.05 | 5.0 | 12.6 | 24.9 | 45.8 | 82.1 | 99.2 | 133.0 | 11.2 | 0.0 |
| Magna | 11/70-12/70 | 37 | 0.0 | 510.9 | 119.0 | 60.0 | 1.67 | 0.0 | 14.3 | 66.5 | 133.5 | 337.0 | 415.2 | 491.1 | 43.2 | 8.1 |
|  | 1/71- 3/71 | 75 | 0.0 | 324.6 | 63.3 | 35.9 | 1.21 | 5.6 | 18.0 | 46.7 | 79.6 | 154.1 | 196.8 | 266.8 | 29.3 | 0.0 |
|  | 4/71- 6/71 | 79 | 0.0 | 531.4 | 76.5 | 44.1 | 1.31 | 5.2 | 28.3 | 64.4 | 95.8 | 150.2 | 174.8 | 341.4 | 40.5 | 1.3 |
|  | 7/71- 9/71 | 83 | 0.0 | 91.4 | 16.2 | 11.3 | 1.33 | 0.0 | 1.1 | 8.9 | 21.1 | 42.7 | 59.8 | 80.6 | 1.2 | 0.0 |
|  | 10/71-12/71 | 88 | 0.6 | 359.3 | 90.1 | 55.8 | 1.24 | 8.3 | 50.6 | 71.5 | 104.5 | 189.4 | 270.1 | 337.8 | 42.0 | 0.0 |
|  | 1/71-12/71 | 325 | 0.0 | 531.4 | 61.8 | 33.8 | 1.39 | 1.4 | 15.2 | 41.7 | 77.7 | 148.2 | 189.1 | 331.1 | 28.3 | 0.3 |
|  | 1/72- 3/72 | 78 | 0.0 | 443.7 | 125.7 | 79.5 | 1.20 | 14.6 | 56.1 | 102.0 | 157.4 | 293.4 | 353.7 | 417.8 | 62.8 | 5.1 |

[a] Concentrations ($\mu g/m^3$) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 80 $\mu g/m^3$.
Standard 2 - National Daily Primary Air Quality Standard, 365 $\mu g/m^3$.

## Table 2.1.A.4. DAILY TOTAL SUSPENDED PARTICULATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - MARCH 1972

| Community | Period of observation | Observations | Concentration, µg/m³ | | | | Geometric standard deviation | Frequency distribution[a] | | | | | | | Observations exceeding standard,[b] % | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Minimum | Maximum | Arithmetic mean | Geometric mean | | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Ogden | 11/70-12/70 | 14 | 23.3 | 167.1 | 84.7 | 72.6 | 0.61 | 32.1 | 48.4 | 83.5 | 107.7 | 140.2 | 156.4 | 165.0 | 50.0 | 0.0 |
| | 1/71- 3/71 | 88 | 18.6 | 276.4 | 92.2 | 84.9 | 0.65 | 33.1 | 53.4 | 69.7 | 115.4 | 177.0 | 226.5 | 267.4 | 47.7 | 2.3 |
| | 4/71- 6/71 | 91 | 15.1 | 222.6 | 62.3 | 55.7 | 0.49 | 29.4 | 47.2 | 58.0 | 73.8 | 96.6 | 117.7 | 139.7 | 28.6 | 0.0 |
| | 7/71- 9/71 | 86 | 33.1 | 166.5 | 71.0 | 67.9 | 0.30 | 47.1 | 60.0 | 66.8 | 77.5 | 99.1 | 113.4 | 127.2 | 36.0 | 0.0 |
| | 10/71-12/71 | 73 | 12.3 | 278.2 | 88.1 | 70.4 | 0.69 | 27.6 | 48.9 | 68.7 | 102.4 | 172.7 | 216.0 | 256.7 | 46.6 | 1.4 |
| | 1/71-12/71 | 338 | 12.3 | 278.2 | 77.9 | 66.5 | 0.56 | 33.0 | 51.7 | 65.3 | 85.0 | 135.0 | 175.5 | 251.9 | 39.3 | 0.9 |
| | 1/72- 3/72 | 91 | 24.8 | 316.6 | 126.3 | 110.1 | 0.56 | 49.5 | 88.5 | 118.1 | 152.6 | 217.7 | 233.2 | 302.1 | 76.9 | 3.3 |
| Salt Lake | 11/70-12/70 | 38 | 20.7 | 230.3 | 85.0 | 70.8 | 0.63 | 33.4 | 47.8 | 72.2 | 115.8 | 157.2 | 168.0 | 208.8 | 50.0 | 0.0 |
| | 1/71- 3/71 | 90 | 20.7 | 238.3 | 98.4 | 84.0 | 0.58 | 40.2 | 61.0 | 86.3 | 115.9 | 181.2 | 207.7 | 240.0 | 58.9 | 0.0 |
| | 4/71- 6/71 | 91 | 25.6 | 197.6 | 64.1 | 59.0 | 0.40 | 34.5 | 49.1 | 58.0 | 71.1 | 96.9 | 109.1 | 177.5 | 24.2 | 0.0 |
| | 7/71- 9/71 | 88 | 28.7 | 130.8 | 67.7 | 65.2 | 0.28 | 44.0 | 59.7 | 66.2 | 75.0 | 87.4 | 99.3 | 127.2 | 30.7 | 0.0 |
| | 10/71-12/71 | 90 | 20.1 | 332.0 | 94.7 | 78.2 | 0.63 | 29.2 | 63.8 | 79.2 | 108.5 | 172.0 | 227.6 | 295.4 | 55.6 | 3.3 |
| | 1/71-12/71 | 359 | 20.1 | 332.0 | 81.2 | 70.9 | 0.51 | 36.2 | 56.4 | 69.5 | 86.5 | 136.0 | 181.1 | 255.4 | 42.3 | 0.8 |
| | 1/72- 3/72 | 87 | 34.1 | 407.3 | 141.3 | 124.7 | 0.51 | 65.5 | 94.6 | 121.0 | 172.7 | 226.5 | 273.5 | 318.9 | 82.8 | 5.7 |
| Kearns | 11/70-12/70 | 35 | 9.4 | 167.0 | 39.8 | 30.7 | 0.71 | 14.3 | 19.3 | 24.0 | 47.9 | 79.5 | 87.8 | 142.7 | 14.3 | 0.0 |
| | 1/71- 3/71 | 80 | 7.2 | 133.5 | 41.9 | 34.9 | 0.64 | 14.2 | 27.3 | 36.4 | 47.9 | 77.9 | 84.5 | 104.6 | 12.5 | 0.0 |
| | 4/71-6/71 | 91 | 0.0 | 157.1 | 44.7 | 39.5 | 0.51 | 22.1 | 28.5 | 37.2 | 47.5 | 76.1 | 98.4 | 141.2 | 11.0 | 0.0 |
| | 7/71- 9/71 | 86 | 15.2 | 125.1 | 45.2 | 42.8 | 0.33 | 28.7 | 38.1 | 44.2 | 49.0 | 58.7 | 75.8 | 89.8 | 5.8 | 0.0 |
| | 10/71-12/71 | 90 | 4.1 | 220.3 | 45.9 | 36.1 | 0.70 | 16.9 | 27.0 | 33.9 | 50.3 | 83.2 | 116.2 | 190.3 | 14.4 | 0.0 |
| | 1/71-12/71 | 347 | 0.0 | 220.3 | 44.5 | 38.2 | 0.57 | 19.9 | 25.9 | 39.7 | 49.0 | 76.5 | 90.6 | 139.1 | 11.0 | 0.0 |
| | 1/72- 3/72 | 87 | 8.4 | 227.9 | 61.9 | 52.6 | 0.57 | 28.9 | 39.1 | 46.2 | 72.5 | 114.8 | 129.8 | 173.0 | 29.9 | 0.0 |
| Magna | 11/70-12/70 | 36 | 6.6 | 191.6 | 61.3 | 40.6 | 0.95 | 11.0 | 22.5 | 37.7 | 70.2 | 152.5 | 174.4 | 187.0 | 30.6 | 0.0 |
| | 1/71- 3/71 | 72 | 13.0 | 460.7 | 82.4 | 62.9 | 0.74 | 22.4 | 45.3 | 65.7 | 92.8 | 136.2 | 195.2 | 346.0 | 41.7 | 4.2 |
| | 4/71- 6/71 | 89 | 17.8 | 243.8 | 54.6 | 47.7 | 0.49 | 26.8 | 35.1 | 46.5 | 57.7 | 88.4 | 108.1 | 210.7 | 15.7 | 0.0 |
| | 7/71- 9/71 | 88 | 7.4 | 182.7 | 55.8 | 51.3 | 0.42 | 32.6 | 43.7 | 51.3 | 62.4 | 88.8 | 102.4 | 129.2 | 13.6 | 0.0 |
| | 10/71-12/71 | 91 | 5.8 | 482.4 | 74.8 | 56.3 | 0.76 | 21.7 | 36.5 | 56.7 | 92.7 | 133.8 | 160.6 | 339.1 | 36.3 | 2.2 |
| | 1/71-12/71 | 340 | 5.8 | 482.4 | 66.2 | 53.9 | 0.62 | 26.2 | 39.0 | 53.4 | 69.5 | 115.1 | 147.7 | 284.0 | 26.2 | 1.5 |
| | 1/72- 3/72 | 90 | 0.0 | 748.4 | 111.0 | 91.2 | 0.62 | 44.0 | 64.8 | 89.7 | 119.9 | 103.0 | 245.7 | 433.5 | 57.8 | 4.4 |

[a] Concentrations (µg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 75 µg/m³.
Standard 2 - National Daily Primary Air Quality Standard, 260 µg/m³.

## Table 2.1.A.5. DAILY SUSPENDED SULFATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standard,[b] % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Ogden | 11/70-12/70 | 14 | 4.1 | 28.5 | 13.1 | 10.9 | 0.65 | 4.9 | 7.2 | 10.1 | 17.3 | 24.7 | 26.6 | 28.1 | 35.7 | 14.3 |
|  | 1/71- 3/71 | 88 | 0.0 | 21.6 | 5.6 | 4.7 | 0.61 | 2.5 | 3.6 | 4.4 | 5.5 | 10.8 | 14.0 | 19.3 | 3.4 | 0.0 |
|  | 4/71-6/71 | 91 | 0.0 | 8.5 | 3.6 | 3.3 | 0.50 | 1.7 | 3.0 | 3.6 | 4.3 | 5.4 | 6.1 | 7.9 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 86 | 2.8 | 12.8 | 5.3 | 5.1 | 0.27 | 3.8 | 4.5 | 5.1 | 5.9 | 6.9 | 7.4 | 9.8 | 0.0 | 0.0 |
|  | 10/71-12/71 | 74 | 3.0 | 34.1 | 8.2 | 7.1 | 0.50 | 3.9 | 5.6 | 6.7 | 8.3 | 12.4 | 19.3 | 31.6 | 8.1 | 4.1 |
|  | 1/71-12/71 | 339 | 0.0 | 34.1 | 5.6 | 4.8 | 0.55 | 2.8 | 3.8 | 4.8 | 5.9 | 8.8 | 11.5 | 21.5 | 2.7 | 0.9 |
|  | 1/72- 3/72 | 91 | 2.6 | 33.1 | 7.9 | 6.7 | 0.53 | 3.9 | 4.9 | 5.9 | 8.2 | 14.7 | 16.5 | 30.1 | 6.6 | 3.3 |
| Salt Lake | 11/70-12/70 | 36 | 0.0 | 66.0 | 15.0 | 11.0 | 0.81 | 4.2 | 6.1 | 10.0 | 17.8 | 30.7 | 38.7 | 62.6 | 34.2 | 18.4 |
|  | 1/71- 3/71 | 90 | 2.3 | 24.9 | 7.2 | 6.2 | 0.53 | 3.3 | 4.6 | 5.9 | 7.8 | 14.2 | 16.5 | 21.8 | 7.8 | 1.1 |
|  | 4/71- 6/71 | 91 | 0.0 | 15.2 | 5.0 | 4.7 | 0.39 | 3.2 | 4.1 | 4.8 | 5.4 | 7.3 | 7.9 | 11.7 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 88 | 1.7 | 18.5 | 5.4 | 5.0 | 0.36 | 3.4 | 4.2 | 5.0 | 5.7 | 7.8 | 9.3 | 13.7 | 1.1 | 0.0 |
|  | 10/71-12/71 | 91 | 2.4 | 54.6 | 11.3 | 9.5 | 0.57 | 4.7 | 7.6 | 9.1 | 11.2 | 21.6 | 25.0 | 40.5 | 16.5 | 6.6 |
|  | 1/71-12/71 | 360 | 0.0 | 54.6 | 7.3 | 6.1 | 0.54 | 3.4 | 4.5 | 5.6 | 7.7 | 12.0 | 17.6 | 28.7 | 6.4 | 1.9 |
|  | 1/72- 3/72 | 87 | 2.4 | 69.6 | 10.2 | 8.0 | 0.63 | 4.0 | 6.1 | 7.2 | 9.5 | 17.8 | 23.1 | 57.9 | 14.9 | 4.6 |
| Kearns | 11/70-12/70 | 35 | 2.8 | 71.2 | 13.8 | 19.7 | 0.83 | 2.9 | 6.8 | 9.3 | 14.6 | 28.1 | 33.2 | 61.7 | 25.7 | 17.1 |
|  | 1/71- 3/71 | 80 | 1.2 | 31.5 | 8.0 | 6.4 | 0.66 | 2.9 | 4.6 | 6.0 | 8.5 | 16.6 | 19.5 | 26.9 | 11.2 | 3.7 |
|  | 4/71- 6/71 | 89 | 0.0 | 15.0 | 5.4 | 5.1 | 0.40 | 3.1 | 4.2 | 5.1 | 5.9 | 8.4 | 9.4 | 14.1 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 86 | 0.0 | 22.5 | 6.0 | 5.4 | 0.47 | 3.4 | 4.2 | 5.2 | 6.5 | 9.6 | 11.5 | 18.7 | 3.5 | 0.0 |
|  | 10/71-12/71 | 88 | 0.6 | 65.9 | 12.0 | 9.3 | 0.71 | 4.8 | 6.6 | 8.7 | 12.6 | 22.8 | 27.6 | 55.5 | 22.7 | 9.1 |
|  | 1/71-12/71 | 344 | 0.0 | 65.9 | 7.8 | 6.4 | 0.62 | 3.2 | 4.7 | 5.9 | 7.7 | 14.8 | 19.0 | 30.5 | 9.3 | 3.2 |
|  | 1/72- 3/72 | 87 | 2.5 | 32.8 | 9.2 | 7.4 | 0.64 | 3.4 | 5.2 | 6.4 | 9.4 | 17.4 | 27.0 | 32.2 | 16.1 | 5.7 |
| Magna | 11/70-12/70 | 36 | 0.8 | 86.4 | 22.0 | 12.8 | 1.12 | 3.7 | 6.5 | 12.4 | 22.5 | 54.9 | 63.4 | 81.6 | 33.3 | 30.6 |
|  | 1/71- 3/71 | 72 | 2.1 | 65.9 | 15.5 | 11.5 | 0.79 | 4.2 | 7.1 | 11.0 | 19.0 | 30.7 | 41.4 | 55.4 | 36.1 | 20.8 |
|  | 4/71- 6/71 | 89 | 2.1 | 28.2 | 9.2 | 8.0 | 0.54 | 4.2 | 6.0 | 8.2 | 10.9 | 15.1 | 17.2 | 23.9 | 9.0 | 1.1 |
|  | 7/71- 9/71 | 88 | 1.1 | 31.5 | 8.9 | 7.1 | 0.68 | 3.5 | 4.5 | 6.7 | 10.8 | 17.9 | 20.7 | 31.4 | 11.4 | 4.5 |
|  | 10/71-12/71 | 92 | 2.2 | 63.9 | 16.5 | 13.2 | 0.70 | 5.7 | 8.4 | 12.7 | 22.4 | 29.8 | 34.7 | 49.8 | 41.3 | 26.1 |
|  | 1/71-12/71 | 341 | 1.1 | 65.9 | 12.4 | 9.6 | 0.72 | 3.8 | 6.2 | 9.2 | 14.0 | 25.7 | 30.7 | 47.6 | 24.0 | 12.9 |
|  | 1/72- 3/72 | 91 | 0.0 | 63.0 | 16.1 | 13.6 | 0.61 | 5.2 | 10.1 | 13.4 | 20.3 | 26.6 | 33.7 | 51.0 | 40.7 | 17.6 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 16 μg/m³.
Standard 2 - National Daily Primary Air Quality Standard, 24 μg/m³.

## Table 2.1.A.6. DAILY NITROGEN DIOXIDE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standard,[b] % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Ogden | 11/70-12/70 | 16 | 30.3 | 88.6 | 59.1 | 56.5 | 0.31 | 40.8 | 48.2 | 52.7 | 74.7 | 81.1 | 83.2 | 87.5 | 0.0 | 0.0 |
|  | 1/71- 3/71 | 73 | 2.8 | 129.0 | 39.6 | 31.1 | 0.78 | 10.7 | 27.0 | 32.4 | 46.1 | 68.7 | 91.7 | 114.8 | 2.7 | 0.0 |
|  | 4/71- 6/71 | 85 | 3.7 | 49.0 | 22.5 | 20.4 | 0.47 | 10.5 | 17.2 | 21.3 | 27.0 | 34.6 | 37.8 | 44.6 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 84 | 7.8 | 82.7 | 35.1 | 30.3 | 0.58 | 12.6 | 23.8 | 33.5 | 41.2 | 59.1 | 67.7 | 78.2 | 0.0 | 0.0 |
|  | 10/71-12/71 | 86 | 15.1 | 117.8 | 52.5 | 47.6 | 0.45 | 25.9 | 38.7 | 48.1 | 61.9 | 79.8 | 102.8 | 117.5 | 5.8 | 0.0 |
|  | 1/71-12/71 | 328 | 2.8 | 129.0 | 37.4 | 31.0 | 0.65 | 13.1 | 23.5 | 32.5 | 44.7 | 67.9 | 79.0 | 115.9 | 2.1 | 0.0 |
|  | 1/72- 3/72 | 86 | 6.0 | 222.1 | 53.2 | 46.5 | 0.53 | 25.9 | 35.4 | 47.0 | 62.1 | 81.1 | 107.6 | 148.5 | 5.8 | 0.0 |
| Salt Lake | 11/70-12/70 | 24 | 10.2 | 202.5 | 65.3 | 57.0 | 0.56 | 31.8 | 50.3 | 59.3 | 68.6 | 92.8 | 108.2 | 181.4 | 8.3 | 0.0 |
|  | 1/71- 3/71 | 73 | 4.5 | 156.3 | 59.2 | 47.9 | 0.72 | 16.5 | 39.0 | 52.7 | 66.8 | 116.4 | 127.0 | 152.7 | 15.1 | 0.0 |
|  | 4/71- 6/71 | 86 | 0.9 | 58.1 | 27.8 | 24.9 | 0.55 | 15.5 | 20.3 | 25.2 | 30.8 | 43.8 | 51.8 | 56.0 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 74 | 3.7 | 65.8 | 32.0 | 27.8 | 0.58 | 13.4 | 23.2 | 29.1 | 40.1 | 55.9 | 59.4 | 63.2 | 0.0 | 0.0 |
|  | 10/71-12/71 | 74 | 8.0 | 188.8 | 45.8 | 37.5 | 0.62 | 16.5 | 30.3 | 37.2 | 46.0 | 71.8 | 122.0 | 173.6 | 6.8 | 0.0 |
|  | 1/71-12/71 | 307 | 0.9 | 188.8 | 40.6 | 33.0 | 0.67 | 15.1 | 24.7 | 34.0 | 44.8 | 68.7 | 102.1 | 156.2 | 5.2 | 0.0 |
|  | 1/72- 3/72 | 87 | 19.7 | 246.5 | 66.0 | 57.3 | 0.52 | 29.4 | 44.0 | 55.8 | 71.3 | 108.9 | 136.6 | 190.9 | 16.1 | 0.0 |
| Kearns | 11/70-12/70 | 27 | 7.6 | 70.6 | 30.6 | 26.1 | 0.58 | 12.5 | 19.2 | 26.2 | 35.3 | 61.3 | 61.9 | 68.4 | 0.0 | 0.0 |
|  | 1/71- 3/71 | 73 | 0.0 | 71.2 | 19.1 | 15.8 | 0.73 | 6.5 | 10.1 | 15.8 | 23.7 | 38.1 | 45.7 | 56.7 | 0.0 | 0.0 |
|  | 4/71- 6/71 | 79 | 2.0 | 47.9 | 12.6 | 10.8 | 0.58 | 5.0 | 8.4 | 11.8 | 13.8 | 19.9 | 25.2 | 35.2 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 79 | 2.1 | 47.1 | 19.6 | 16.7 | 0.62 | 7.0 | 13.0 | 18.2 | 24.0 | 34.5 | 35.9 | 43.8 | 0.0 | 0.0 |
|  | 10/71-12/71 | 87 | 6.9 | 201.8 | 36.3 | 28.1 | 0.68 | 12.4 | 18.3 | 25.9 | 37.0 | 62.9 | 96.3 | 171.1 | 4.6 | 0.0 |
|  | 1/71-12/71 | 318 | 0.0 | 201.8 | 22.3 | 17.0 | 0.74 | 6.7 | 11.4 | 17.2 | 24.5 | 41.6 | 51.0 | 116.2 | 1.3 | 0.0 |
|  | 1/72- 3/72 | 80 | 5.8 | 81.8 | 27.8 | 24.6 | 0.49 | 13.9 | 20.3 | 24.1 | 29.4 | 45.6 | 58.0 | 75.3 | 0.0 | 0.0 |
| Magna | 11/70-12/70 | 26 | 2.5 | 129.8 | 36.6 | 27.5 | 0.84 | 10.9 | 20.0 | 30.3 | 44.3 | 58.1 | 86.8 | 121.2 | 3.8 | 0.0 |
|  | 1/71- 3/71 | 74 | 0.0 | 82.5 | 22.4 | 17.7 | 0.81 | 5.9 | 11.8 | 16.9 | 26.1 | 47.0 | 56.4 | 73.8 | 0.0 | 0.0 |
|  | 4/71- 6/71 | 80 | 0.0 | 108.6 | 12.9 | 9.3 | 0.84 | 2.6 | 6.5 | 9.2 | 14.3 | 23.2 | 28.2 | 61.5 | 1.2 | 0.0 |
|  | 7/71- 9/71 | 83 | 0.0 | 53.8 | 15.3 | 12.4 | 0.76 | 2.8 | 8.0 | 13.3 | 18.0 | 32.4 | 38.5 | 48.1 | 0.0 | 0.0 |
|  | 10/71-12/71 | 87 | 5.2 | 134.3 | 31.8 | 25.8 | 0.64 | 11.6 | 18.6 | 24.6 | 34.0 | 56.6 | 86.5 | 115.3 | 2.3 | 0.0 |
|  | 1/71-12/71 | 324 | 0.0 | 134.3 | 20.8 | 15.3 | 0.85 | 4.4 | 9.9 | 15.6 | 23.2 | 42.8 | 54.1 | 99.4 | 0.9 | 0.0 |
|  | 1/72- 3/72 | 78 | 2.8 | 118.9 | 32.4 | 25.7 | 0.72 | 10.5 | 19.4 | 26.4 | 36.5 | 57.9 | 73.2 | 114.2 | 3.8 | 0.0 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 100 μg/m³.
Standard 2 - National Daily Primary Air Quality Standard, 350 μg/m³.

## Table 2.1.A.7. DAILY SUSPENDED NITRATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standard,[b] % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Ogden | 11/70-12/70 | 14 | 1.1 | 16.8 | 6.2 | 4.3 | 0.94 | 1.2 | 2.0 | 5.5 | 7.4 | 14.1 | 16.1 | 16.7 | 64.3 | 28.6 |
|  | 1/71- 3/71 | 88 | 0.2 | 11.5 | 2.3 | 1.6 | 0.84 | 0.6 | 1.0 | 1.5 | 2.6 | 4.7 | 7.1 | 8.8 | 30.7 | 4.5 |
|  | 4/71- 6/71 | 91 | 0.3 | 3.8 | 1.6 | 1.5 | 0.49 | 0.8 | 1.2 | 1.5 | 1.9 | 2.6 | 3.0 | 3.7 | 13.2 | 0.0 |
|  | 7/71- 9/71 | 86 | 0.5 | 5.2 | 2.6 | 2.4 | 0.43 | 1.4 | 2.0 | 2.6 | 3.2 | 3.9 | 4.2 | 4.8 | 57.0 | 0.0 |
|  | 10/71-12/71 | 74 | 0.6 | 25.1 | 4.7 | 3.2 | 0.85 | 1.2 | 1.8 | 3.2 | 5.0 | 10.5 | 13.7 | 21.8 | 58.1 | 17.6 |
|  | 1/71-12/71 | 339 | 0.2 | 25.1 | 2.7 | 2.0 | 0.74 | 0.8 | 1.4 | 1.9 | 2.9 | 4.8 | 7.4 | 13.8 | 38.6 | 5.0 |
|  | 1/72- 3/72 | 91 | 0.0 | 49.3 | 4.6 | 2.1 | 1.32 | 0.6 | 1.2 | 1.9 | 3.5 | 11.2 | 17.0 | 35.4 | 39.6 | 15.4 |
| Salt Lake | 11/70-12/70 | 38 | 0.3 | 12.6 | 3.0 | 2.0 | 0.95 | 0.6 | 1.3 | 1.9 | 3.5 | 7.6 | 8.0 | 11.2 | 34.2 | 10.5 |
|  | 1/71- 3/71 | 90 | 0.2 | 12.0 | 2.7 | 1.9 | 0.87 | 0.7 | 1.1 | 1.4 | 3.1 | 6.6 | 8.2 | 10.1 | 34.4 | 8.9 |
|  | 4/71- 6/71 | 91 | 0.4 | 6.3 | 2.2 | 1.9 | 0.60 | 0.8 | 1.5 | 2.0 | 2.6 | 4.1 | 4.6 | 5.5 | 31.9 | 0.0 |
|  | 7/71- 9/71 | 88 | 0.4 | 5.9 | 2.9 | 2.6 | 0.49 | 1.4 | 2.2 | 2.8 | 3.4 | 4.3 | 4.7 | 5.4 | 56.8 | 0.0 |
|  | 10/71-12/71 | 91 | 0.5 | 34.0 | 5.6 | 3.6 | 0.90 | 1.2 | 2.2 | 3.5 | 5.9 | 12.3 | 19.9 | 26.8 | 65.9 | 16.6 |
|  | 1/71-12/71 | 360 | 0.2 | 34.0 | 3.3 | 2.4 | 0.79 | 0.9 | 1.6 | 2.4 | 3.5 | 6.1 | 8.4 | 21.4 | 47.2 | 6.4 |
|  | 1/72- 3/72 | 87 | 0.3 | 52.2 | 6.0 | 2.9 | 1.14 | 0.9 | 1.5 | 2.3 | 4.2 | 16.9 | 27.4 | 45.4 | 46.0 | 20.7 |
| Kearns | 11/70-12/70 | 35 | 0.0 | 7.5 | 1.4 | 0.9 | 0.94 | 0.4 | 0.6 | 0.9 | 1.7 | 2.0 | 4.4 | 7.1 | 8.6 | 2.9 |
|  | 1/71- 3/71 | 80 | 0.0 | 5.6 | 1.6 | 1.1 | 0.97 | 0.3 | 0.8 | 1.2 | 1.8 | 3.8 | 4.8 | 5.3 | 21.2 | 0.0 |
|  | 4/71- 6/71 | 90 | 0.3 | 4.8 | 1.6 | 1.4 | 0.52 | 0.7 | 1.1 | 1.4 | 1.8 | 2.7 | 3.0 | 3.6 | 14.4 | 0.0 |
|  | 7/71- 9/71 | 86 | 0.4 | 4.3 | 2.1 | 1.9 | 0.45 | 1.1 | 1.7 | 2.0 | 2.4 | 3.1 | 3.5 | 4.2 | 29.1 | 0.0 |
|  | 10/71-12/71 | 88 | 0.2 | 47.1 | 4.3 | 2.5 | 1.05 | 0.7 | 1.6 | 2.6 | 4.4 | 9.4 | 11.8 | 19.6 | 52.3 | 15.9 |
|  | 1/71-12/71 | 344 | 0.0 | 47.1 | 2.4 | 1.7 | 0.85 | 0.6 | 1.2 | 1.7 | 2.4 | 4.4 | 6.2 | 12.1 | 29.4 | 4.1 |
|  | 1/72- 3/72 | 87 | 0.0 | 37.6 | 4.8 | 2.1 | 1.29 | 0.5 | 0.9 | 1.6 | 3.1 | 15.3 | 24.5 | 32.9 | 36.8 | 18.4 |
| Magna | 11/70-12/70 | 36 | 0.0 | 8.9 | 1.4 | 0.8 | 1.08 | 0.3 | 0.4 | 0.8 | 1.5 | 2.5 | 4.1 | 8.0 | 11.1 | 2.8 |
|  | 1/71- 3/71 | 72 | 0.0 | 10.9 | 1.7 | 0.9 | 1.19 | 0.2 | 0.5 | 1.0 | 1.7 | 4.1 | 6.2 | 9.6 | 19.4 | 4.2 |
|  | 4/71- 6/71 | 89 | 0.1 | 4.3 | 1.2 | 1.0 | 0.65 | 0.5 | 0.8 | 1.1 | 1.5 | 2.0 | 2.3 | 3.6 | 4.5 | 0.0 |
|  | 7/71- 9/71 | 88 | 0.0 | 4.0 | 1.7 | 1.5 | 0.60 | 0.9 | 1.3 | 1.7 | 2.0 | 2.5 | 3.0 | 3.9 | 10.2 | 0.0 |
|  | 10/71-12/71 | 92 | 0.0 | 20.3 | 3.3 | 1.9 | 1.09 | 0.4 | 1.2 | 2.1 | 3.0 | 9.6 | 11.5 | 17.3 | 38.0 | 13.0 |
|  | 1/71-12/71 | 341 | 0.0 | 20.3 | 2.0 | 1.3 | 0.95 | 0.4 | 1.0 | 1.4 | 2.0 | 3.6 | 5.7 | 12.0 | 18.2 | 4.4 |
|  | 1/72- 3/72 | 91 | 0.0 | 32.6 | 4.2 | 1.6 | 1.44 | 0.2 | 0.8 | 1.8 | 2.4 | 13.2 | 20.3 | 26.7 | 29.7 | 16.5 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - tentative annual air quality standard, 2.5 μg/m³.
Standard 2 - tentative daily air quality standard, 7.5 μg/m³.

### Table 2.1.A.8. MONTHLY TOTAL DUSTFALL RATES (g/m²/mo) FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - FEBRUARY 1972

| Month | Ogden | Salt Lake City | Kearns | Magna |
|---|---|---|---|---|
| Nov. 70 | 3.57 | 2.34 | 1.11 | 2.86 |
| Dec. | 1.16 | 0.77 | 0.09 | 1.65 |
| Jan. 71 | 5.29 | 4.84 | 1.19 | 2.45 |
| Feb. | 8.10 | 4.13 | 2.72 | 2.72 |
| March | 7.39 | 7.00 | 4.76 | 19.00 |
| April | 7.58 | – | 4.29 | 2.64 |
| May | 4.51 | 6.50 | 4.29 | – |
| June | 3.19 | 0.60 | 2.69 | 3.89 |
| July | 2.53 | – | 1.99 | 2.60 |
| Aug. | 3.40 | 6.89 | 2.07 | 2.50 |
| Sept. | – | 1.24 | 0.74 | 4.82 |
| Oct. | 3.62 | 0.59 | 0.96 | 3.14 |
| Nov. | 1.45 | 2.00 | 1.11 | 3.29 |
| Dec. | 0.73 | 1.81 | – | 3.70 |
| Jan. 72 | 1.68 | – | 1.38 | 3.33 |
| Feb. | 4.02 | 3.52 | 2.98 | 3.44 |

### Table 2.1.A.9. MONTHLY CADMIUM CONCENTRATIONS (mg/m²/mo) IN DUSTFALL FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - FEBRUARY 1972

| Month | Ogden | Salt Lake City | Kearns | Magna |
|---|---|---|---|---|
| Nov. 70 | 0.06 | 0.12 | 0.00 | 0.17 |
| Dec. | 0.05 | 0.00 | 0.00 | 0.00 |
| Jan: 71 | 0.14 | 0.07 | 0.05 | 0.05 |
| Feb. | 0.08 | 0.07 | 0.15 | 0.00 |
| March | 0.07 | 0.10 | 0.09 | 0.19 |
| April | 0.15 | 0.12 | 0.39 | 0.20 |
| May | 0.18 | 0.19 | 0.19 | – |
| June | 0.03 | 0.03 | 0.63 | 0.14 |
| July | 0.07 | – | 0.05 | 0.05 |
| Aug. | 0.04 | 0.15 | 0.07 | 0.07 |
| Sept. | – | 0.08 | 0.02 | 0.16 |
| Oct. | 0.09 | 0.28 | 0.11 | 0.23 |
| Nov. | 0.02 | 0.04 | 0.14 | 0.07 |
| Dec. | 0.01 | – | 0.02 | 0.04 |
| Jan. 72 | 0.03 | 0.08 | 0.05 | 0.05 |
| Feb. | 0.02 | 0.03 | – | 0.02 |

### Table 2.1.A.10. MONTHLY LEAD CONCENTRATIONS (mg/m²/mo) IN DUSTFALL FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - FEBRUARY 1972

| Month | Ogden | Salt Lake City | Kearns | Magna |
|---|---|---|---|---|
| Nov. 70 | 3.45 | 5.71 | 1.19 | 22.98 |
| Dec. | 3.58 | 2.23 | 0.46 | 1.84 |
| Jan. 71 | 12.86 | 12.02 | 3.21 | 3.21 |
| Feb. | 8.74 | 11.71 | 4.36 | 1.95 |
| March | 10.61 | 13.52 | 3.97 | 7.75 |
| April | 8.95 | 7.54 | 4.25 | 6.08 |
| May | 6.98 | 17.94 | 6.54 | – |
| June | 4.60 | 0.57 | 6.98 | 4.96 |
| July | 3.90 | – | 1.38 | 1.26 |
| Aug. | 4.77 | 4.61 | 4.43 | 5.46 |
| Sept. | – | 5.12 | 0.60 | 4.02 |
| Oct. | 7.43 | 1.77 | 7.20 | 4.82 |
| Nov. | 2.41 | 7.29 | 2.18 | 2.41 |
| Dec. | 2.48 | – | 1.56 | 2.02 |
| Jan. 72 | 4.02 | 10.31 | 1.61 | 2.30 |
| Feb. | 7.23 | 8.49 | 2.07 | 2.53 |

### Table 2.1.A.11. MONTHLY ZINC CONCENTRATION (mg/m²/mo) IN DUSTFALL FOR UTAH CHESS COMMUNITIES, NOVEMBER 1970 - FEBRUARY 1972

| Month | Ogden | Salt Lake City | Kearns | Magna |
|---|---|---|---|---|
| Nov. 70 | 1.43 | 2.74 | 0.24 | 3.21 |
| Dec. | 0.92 | 1.70 | 0.64 | 0.37 |
| Jan. 71 | 4.07 | 5.48 | 1.72 | 0.92 |
| Feb. | 3.86 | 3.79 | 3.10 | 0.46 |
| March | 4.82 | 6.43 | 3.88 | 6.52 |
| April | 7.81 | 4.88 | 5.28 | 5.05 |
| May | 5.99 | 6.32 | 3.99 | – |
| June | 2.61 | 1.04 | 7.62 | 4.68 |
| July | 5.28 | – | 2.64 | 2.98 |
| Aug. | 4.21 | 0.62 | 3.99 | 3.54 |
| Sept. | – | 6.13 | 0.90 | 28.33 |
| Oct. | 5.76 | 1.11 | 3.55 | 7.69 |
| Nov. | 1.26 | 3.00 | 3.79 | 3.33 |
| Dec. | 1.19 | – | 0.73 | 2.76 |
| Jan. 72 | 1.95 | 6.32 | 5.06 | 2.76 |
| Feb. | 3.10 | 4.71 | 5.51 | 3.10 |

Table 2.1.A.12. CORRELATION MATRIX FOR POLLUTANTS AND AIR MONITORING SITES FOR UTAH CHESS COMMUNITIES, JANUARY - DECEMBER 1971[a]

| Pollutant-site | Correlation coefficient | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | TSP-11 | SN-11 | SS-11 | $SO_2$-11 | $NO_2$-11 | TSP-21 | SN-21 | SS-21 | $SO_2$-21 | $NO_2$-21 | TSP-31 | SN-31 | SS-31 | $SO_2$-31 | $NO_2$-31 | TSP-41 | SN-41 | SS-41 | $SO_2$-41 | $NO_2$-41 |
| TSP-11 | 1.00 | 0.60 | 0.48 | 0.39 | 0.48 | 0.81 | 0.48 | 0.41 | 0.27 | 0.47 | 0.49 | 0.35 | 0.34 | 0.22 | 0.41 | 0.44 | 0.40 | 0.44 | 0.33 | 0.48 |
| SN-11 | 0.56 | 1.00 | 0.83 | 0.46 | 0.59 | 0.67 | 0.87 | 0.79 | 0.33 | 0.49 | 0.49 | 0.69 | 0.65 | 0.35 | 0.73 | 0.39 | 0.63 | 0.56 | 0.36 | 0.59 |
| SS-11 | 0.48 | 0.83 | 1.00 | 0.46 | 0.64 | 0.55 | 0.71 | 0.79 | 0.26 | 0.45 | 0.36 | 0.58 | 0.57 | 0.20 | 0.71 | 0.30 | 0.53 | 0.48 | 0.29 | 0.56 |
| $SO_2$-11 | 0.39 | 0.47 | 0.46 | 1.00 | 0.48 | 0.34 | 0.33 | 0.31 | 0.28 | 0.20 | 0.14 | 0.20 | 0.19 | 0.19 | 0.31 | 0.12 | 0.29 | 0.28 | 0.41 | 0.27 |
| $NO_2$-11 | 0.48 | 0.59 | 0.64 | 0.48 | 1.00 | 0.50 | 0.49 | 0.51 | 0.35 | 0.55 | 0.23 | 0.44 | 0.47 | 0.28 | 0.58 | 0.28 | 0.44 | 0.51 | 0.36 | 0.60 |
| TSP-21 | 0.81 | 0.67 | 0.55 | 0.34 | 0.50 | 1.00 | 0.64 | 0.57 | 0.29 | 0.60 | 0.62 | 0.53 | 0.51 | 0.31 | 0.55 | 0.51 | 0.47 | 0.54 | 0.35 | 0.56 |
| SN-21 | 0.48 | 0.88 | 0.71 | 0.33 | 0.49 | 0.64 | 1.00 | 0.80 | 0.31 | 0.46 | 0.54 | 0.76 | 0.69 | 0.40 | 0.76 | 0.39 | 0.60 | 0.52 | 0.31 | 0.54 |
| SS-21 | 0.41 | 0.79 | 0.79 | 0.31 | 0.51 | 0.57 | 0.80 | 1.00 | 0.31 | 0.41 | 0.40 | 0.67 | 0.72 | 0.36 | 0.72 | 0.36 | 0.53 | 0.56 | 0.36 | 0.54 |
| $SO_2$-21 | 0.27 | 0.33 | 0.26 | 0.28 | 0.35 | 0.29 | 0.31 | 0.31 | 1.00 | 0.30 | 0.19 | 0.24 | 0.32 | 0.22 | 0.33 | 0.13 | 0.30 | 0.23 | 0.21 | 0.30 |
| $NO_2$-21 | 0.47 | 0.49 | 0.45 | 0.20 | 0.55 | 0.60 | 0.46 | 0.41 | 0.30 | 1.00 | 0.30 | 0.33 | 0.45 | 0.30 | 0.54 | 0.30 | 0.46 | 0.52 | 0.27 | 0.63 |
| TSP-31 | 0.49 | 0.49 | 0.36 | 0.14 | 0.23 | 0.62 | 0.54 | 0.40 | 0.19 | 0.30 | 1.00 | 0.49 | 0.53 | 0.36 | 0.51 | 0.66 | 0.40 | 0.37 | 0.19 | 0.39 |
| SN-31 | 0.35 | 0.69 | 0.56 | 0.20 | 0.44 | 0.53 | 0.76 | 0.67 | 0.24 | 0.33 | 0.49 | 1.00 | 0.69 | 0.24 | 0.71 | 0.30 | 0.50 | 0.35 | 0.18 | 0.41 |
| SS-31 | 0.34 | 0.65 | 0.57 | 0.19 | 0.47 | 0.51 | 0.69 | 0.72 | 0.32 | 0.45 | 0.53 | 0.69 | 1.00 | 0.58 | 0.70 | 0.41 | 0.44 | 0.63 | 0.44 | 0.53 |
| $SO_2$-31 | 0.22 | 0.35 | 0.20 | 0.19 | 0.28 | 0.31 | 0.40 | 0.36 | 0.22 | 0.30 | 0.36 | 0.24 | 0.58 | 1.00 | 0.39 | 0.40 | 0.44 | 0.59 | 0.65 | 0.43 |
| $NO_2$-31 | 0.41 | 0.73 | 0.71 | 0.31 | 0.58 | 0.55 | 0.76 | 0.72 | 0.33 | 0.54 | 0.51 | 0.71 | 0.70 | 0.39 | 1.00 | 0.37 | 0.59 | 0.49 | 0.27 | 0.70 |
| TSP-41 | 0.44 | 0.34 | 0.30 | 0.12 | 0.28 | 0.51 | 0.39 | 0.36 | 0.13 | 0.30 | 0.66 | 0.30 | 0.41 | 0.40 | 0.37 | 1.00 | 0.39 | 0.50 | 0.32 | 0.47 |
| SN-41 | 0.40 | 0.63 | 0.53 | 0.29 | 0.44 | 0.47 | 0.60 | 0.53 | 0.30 | 0.46 | 0.40 | 0.50 | 0.44 | 0.44 | 0.59 | 0.39 | 1.00 | 0.50 | 0.33 | 0.58 |
| SS-41 | 0.44 | 0.56 | 0.48 | 0.28 | 0.51 | 0.54 | 0.52 | 0.56 | 0.23 | 0.52 | 0.37 | 0.35 | 0.63 | 0.59 | 0.49 | 0.50 | 0.50 | 1.00 | 0.67 | 0.63 |
| $SO_2$-41 | 0.33 | 0.36 | 0.29 | 0.41 | 0.36 | 0.34 | 0.31 | 0.36 | 0.21 | 0.27 | 0.19 | 0.18 | 0.44 | 0.65 | 0.27 | 0.32 | 0.33 | 0.67 | 1.00 | 0.45 |
| $NO_2$-41 | 0.48 | 0.59 | 0.56 | 0.27 | 0.60 | 0.56 | 0.54 | 0.54 | 0.30 | 0.63 | 0.39 | 0.41 | 0.53 | 0.43 | 0.70 | 0.47 | 0.58 | 0.63 | 0.45 | 1.00 |

[a] Site identification: 11 - Ogden, 21 - Salt Lake City, 31 - Kearns, 41 - Magna. Pollutant identification: TSP - total suspended particulate, SN - suspended nitrates, SS - suspended sulfates, $SO_2$ - sulfur dioxide, and $NO_2$ - nitrogen dioxide.

Table 2.1.A.13. CORRELATION MATRIX FOR POLLUTANTS AND AIR MONITORING SITES FOR UTAH CHESS COMMUNITIES, JANUARY - MARCH 1972[a]

| Pollutant-site | Correlation coefficient | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | TSP-11 | SN-11 | SS-11 | $SO_2$-11 | $NO_2$-11 | TSP-21 | SN-21 | SS-21 | $SO_2$-21 | $NO_2$-21 | TSP-31 | SN-31 | SS-31 | $SO_2$-31 | $NO_2$-31 | TSP-41 | SN-41 | SS-41 | $SO_2$-41 | $NO_2$-41 |
| TSP-11 | 1.00 | 0.31 | 0.35 | 0.31 | 0.26 | 0.72 | 0.28 | 0.23 | 0.17 | 0.06 | 0.53 | 0.29 | 0.33 | 0.28 | 0.22 | 0.13 | 0.31 | 0.34 | 0.30 | 0.29 |
| SN-11 | 0.31 | 1.00 | 0.77 | 0.43 | 0.43 | 0.42 | 0.63 | 0.49 | 0.53 | 0.38 | 0.41 | 0.65 | 0.64 | 0.48 | 0.62 | 0.14 | 0.62 | 0.48 | 0.24 | 0.39 |
| SS-11 | 0.35 | 0.77 | 1.00 | 0.43 | 0.34 | 0.58 | 0.83 | 0.81 | 0.41 | 0.29 | 0.47 | 0.76 | 0.70 | 0.47 | 0.62 | 0.15 | 0.68 | 0.65 | 0.38 | 0.34 |
| $SO_2$-11 | 0.31 | 0.43 | 0.43 | 1.00 | 0.31 | 0.25 | 0.25 | 0.24 | 0.17 | 0.19 | 0.16 | 0.21 | 0.20 | 0.07 | 0.27 | 0.03 | 0.35 | 0.20 | 0.05 | 0.33 |
| $NO_2$-11 | 0.26 | 0.43 | 0.34 | 0.31 | 1.00 | 0.37 | 0.31 | 0.27 | 0.19 | 0.71 | 0.12 | 0.25 | 0.21 | 0.18 | 0.35 | 0.07 | 0.27 | 0.22 | 0.13 | 0.26 |
| TSP-21 | 0.72 | 0.42 | 0.58 | 0.25 | 0.37 | 1.00 | 0.66 | 0.62 | 0.51 | 0.39 | 0.67 | 0.60 | 0.56 | 0.49 | 0.46 | 0.27 | 0.57 | 0.55 | 0.33 | 0.42 |
| SN-21 | 0.28 | 0.63 | 0.83 | 0.25 | 0.31 | 0.66 | 1.00 | 0.94 | 0.58 | 0.44 | 0.51 | 0.91 | 0.78 | 0.52 | 0.71 | 0.24 | 0.76 | 0.72 | 0.43 | 0.53 |
| SS-21 | 0.23 | 0.49 | 0.81 | 0.24 | 0.27 | 0.62 | 0.94 | 1.00 | 0.46 | 0.39 | 0.47 | 0.83 | 0.73 | 0.51 | 0.63 | 0.27 | 0.68 | 0.69 | 0.43 | 0.52 |
| $SO_2$-21 | 0.17 | 0.53 | 0.41 | 0.17 | 0.19 | 0.51 | 0.58 | 0.46 | 1.00 | 0.43 | 0.43 | 0.62 | 0.59 | 0.51 | 0.58 | 0.32 | 0.49 | 0.43 | 0.30 | 0.32 |
| $NO_2$-11 | 0.06 | 0.39 | 0.29 | 0.19 | 0.71 | 0.39 | 0.44 | 0.39 | 0.43 | 1.00 | 0.20 | 0.35 | 0.37 | 0.29 | 0.37 | 0.34 | 0.41 | 0.31 | 0.05 | 0.32 |
| TSP-31 | 0.53 | 0.41 | 0.47 | 0.16 | 0.12 | 0.67 | 0.51 | 0.47 | 0.43 | 0.20 | 1.00 | 0.48 | 0.46 | 0.37 | 0.36 | 0.43 | 0.41 | 0.45 | 0.24 | 0.31 |
| SN-31 | 0.29 | 0.65 | 0.76 | 0.21 | 0.25 | 0.60 | 0.91 | 0.83 | 0.62 | 0.35 | 0.48 | 1.00 | 0.81 | 0.46 | 0.75 | 0.20 | 0.78 | 0.65 | 0.35 | 0.49 |
| SS-31 | 0.33 | 0.64 | 0.70 | 0.20 | 0.21 | 0.56 | 0.78 | 0.73 | 0.59 | 0.37 | 0.46 | 0.81 | 1.00 | 0.59 | 0.66 | 0.23 | 0.66 | 0.70 | 0.42 | 0.67 |
| $SO_2$-31 | 0.28 | 0.48 | 0.47 | 0.07 | 0.18 | 0.49 | 0.52 | 0.51 | 0.51 | 0.29 | 0.37 | 0.46 | 0.59 | 1.00 | 0.53 | 0.11 | 0.46 | 0.49 | 0.54 | 0.40 |
| $NO_2$-31 | 0.22 | 0.62 | 0.62 | 0.27 | 0.35 | 0.46 | 0.71 | 0.63 | 0.58 | 0.37 | 0.36 | 0.75 | 0.66 | 0.53 | 1.00 | 0.21 | 0.66 | 0.61 | 0.29 | 0.67 |
| TSP-41 | 0.13 | 0.14 | 0.15 | 0.03 | 0.07 | 0.27 | 0.24 | 0.27 | 0.32 | 0.34 | 0.43 | 0.20 | 0.23 | 0.11 | 0.21 | 1.00 | 0.18 | 0.32 | 0.13 | 0.23 |
| SN-41 | 0.31 | 0.62 | 0.68 | 0.35 | 0.27 | 0.57 | 0.76 | 0.68 | 0.49 | 0.41 | 0.41 | 0.78 | 0.66 | 0.46 | 0.66 | 0.18 | 1.00 | 0.60 | 0.20 | 0.43 |
| SS-41 | 0.34 | 0.48 | 0.65 | 0.20 | 0.22 | 0.55 | 0.72 | 0.69 | 0.43 | 0.31 | 0.45 | 0.65 | 0.70 | 0.49 | 0.61 | 0.32 | 0.60 | 1.00 | 0.68 | 0.71 |
| $SO_2$-41 | 0.30 | 0.24 | 0.38 | 0.05 | 0.13 | 0.33 | 0.43 | 0.43 | 0.30 | 0.05 | 0.24 | 0.35 | 0.42 | 0.54 | 0.29 | 0.13 | 0.20 | 0.68 | 1.00 | 0.42 |
| $NO_2$-41 | 0.26 | 0.39 | 0.55 | 0.22 | 0.26 | 0.42 | 0.54 | 0.52 | 0.33 | 0.33 | 0.31 | 0.50 | 0.68 | 0.41 | 0.67 | 0.23 | 0.44 | 0.72 | 0.42 | 1.00 |

[a] Site identification: 11-Ogden, 21-Salt Lake City, 31-Kearns, 41-Magna. Pollutant identification: TSP-total suspended particulate, SN-suspended nitrates, SS-suspended sulfates, $SO_2$-sulfur dioxide, $NO_2$-nitrogen dioxide.

### Table 2.1.A.14. EMISSIONS FROM SMELTER AND ESTIMATED SULFUR DIOXIDE ($SO_2$) AND SUSPENDED SULFATE (SS) CONCENTRATIONS FOR MAGNA AND KEARNS 1940 - 1971

| Year | $SO_2$ emissions from smelter, tons/day | Concentrations,[a] $\mu g/m^3$ | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Diffusion model Magna $SO_2$ | CHESS[b] | | | | Utah State Division of Health, $SO_2$[d] | |
| | | | Magna | | Kearns[c] | | | |
| | | | $SO_2$ | SS | $SO_2$ | SS | Magna | Kearns |
| 1971 | 193 | 41 | 61.8 | 12.4 | 21.7 | 7.8 | 107.4 | 34.1 |
| 1970 | 261 | 56 | (84) | (16.7) | (29) | (10.5) | 120.5 | (38) |
| 1969 | 322 | 69 | (103) | (21.0) | (36) | (13.2) | (162) | (51) |
| 1968 | 281 | 60 | (90) | (18.1) | (32) | 11.4 | (141) | (45) |
| 1967 | 276 | 59 | (88) | (17.7) | (31) | 11.2 | (139) | (44) |
| 1966 | 301 | 64 | (96) | (19.3) | (34) | 12.2 | (150) | (48) |
| 1965 | 302 | 65 | (97) | 19.3 | (34) | 12.2 | (153) | (49) |
| 1964 | 311 | 67 | (100) | 20.0 | (35) | 12.6 | (157) | (50) |
| 1963 | 260 | 56 | (83) | 16.7 | (29) | 10.5 | (132) | (42) |
| 1962 | 349 | 75 | (112) | 22.4 | (39) | 14.1 | (176) | (56) |
| 1961 | 331 | 71 | (106) | 21.3 | (37) | 13.5 | (167) | (53) |
| 1960 | 247 | 53 | (79) | 15.9 | (28) | 10.0 | (125) | (40) |
| 1959 | 224 | 48 | (72) | 14.4 | (25) | 9.0 | (113) | (36) |
| 1958 | 202 | 43 | (65) | 13.0 | (23) | 8.2 | (101) | (32) |
| 1957 | 250 | 54 | (80) | 16.1 | (28) | 10.1 | (127) | (40) |
| 1956 | 448 | 96 | (143) | 28.8 | (50) | 18.1 | (226) | (72) |
| 1955 | 445 | 95 | (142) | 28.6 | (50) | 18.0 | (223) | (71) |
| 1954 | 489 | 105 | (157) | 31.4 | (55) | 19.7 | (247) | (78) |
| 1953 | 557 | 119 | (178) | 35.8 | (63) | 22.5 | (280) | (89) |
| 1952 | 581 | 124 | (186) | 37.3 | | | (291) | |
| 1951 | 600 | 128 | (192) | 38.6 | | | (301) | |
| 1950 | 649 | 139 | (208) | 41.7 | | | (327) | |
| 1949 | 554 | 119 | (177) | 35.6 | | | (280) | |
| 1948 | 614 | 131 | (197) | 39.4 | | | (308) | |
| 1947 | 616 | 132 | (197) | 39.6 | | | (310) | |
| 1946 | 475 | 102 | (152) | 30.5 | | | (240) | |
| 1945 | 575 | 123 | (184) | 37.0 | | | (289) | |
| 1944 | 734 | 157 | (235) | 47.1 | | | (369) | |
| 1943 | 888 | 190 | (284) | 57.0 | | | (446) | |
| 1942 | 892 | 191 | (286) | 57.3 | | | (449) | |
| 1941 | 976 | 209 | (313) | 62.7 | | | (491) | |
| 1940 | 970 | 208 | (310) | 62.4 | | | (489) | |

[a] Estimated values in parentheses.

[b] West-Gaeke method.

[c] Kearns was not established until 1953.

[d] Conductometric method; subject to interference.

Table 2.1.A.15. ANNUAL PRODUCTION ($10^3$ tons) OF COPPER BY THE SMELTER AT GARFIELD, UTAH, 1940-1971[16]

| Year | Copper production | Year | Copper production |
|---|---|---|---|
| 1971 | 260 | 1955 | 233 |
| 1970 | 296 | 1954 | 212 |
| 1969 | 297 | 1953 | 269 |
| 1968 | 228 | 1952 | 283 |
| 1967 | 169 | 1951 | 271 |
| 1966 | 265 | 1950 | 279 |
| 1965 | 259 | 1949 | 197 |
| 1964 | 200 | 1948 | 227 |
| 1963 | 203 | 1947 | 267 |
| 1962 | 218 | 1946 | 114 |
| 1961 | 213 | 1945 | 226 |
| 1960 | 219 | 1944 | 302 |
| 1959 | 145 | 1943 | 326 |
| 1958 | 189 | 1942 | 318 |
| 1957 | 238 | 1941 | 271 |
| 1956 | 251 | 1940 | 249 |

## Table 2.1.A.16. POLLUTANT SUMMARY FOR UTAH CHESS COMMUNITIES, 1940 - 1971[a]

| | Ogden | | | Salt Lake City | | | Kearns | | | Magna | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Year | $SO_2$ | TSP | SS | $SO_2$ | TSP | SS | $SO_2$ | TSP | SS | $SO_2$ | TSP | SS |
| 1971 | 8.0[b] | 78[b] | 5.6[b] | 15.3[b] | 81.2[b] | 7.3[b] | 21.7 | 44[b] | 7.8[b] | 61.8[b] | 66[b] | 12.4[b] |
| 1970 | | 78[c] | 4.4[d] | 8.0[d] | 88.0[d] | 5.2[d] | (29) | (50) | (8.3) | (84) | (75) | (14.2) |
| 1969 | | 95[c] | -- | 28.0[d] | 90.0[d] | (5.4) | (36) | (50) | (9.0) | (103) | (75) | (15.9) |
| 1968 | | 91[c] | 2.9[d] | 17.0[d] | 76.0[d] | 5.2[d] | (32) | (39) | (8.6) | (90) | (58) | (14.8) |
| 1967 | | 103[c] | -- | 17.0[d] | 77.0[d] | 3.6[d] | (31) | (28) | (8.5) | (88) | (43) | (14.6) |
| 1966 | | 94[c] | 4.8[d] | 20.0[d] | 93.0[d] | 6.9[d] | (34) | (45) | (8.8) | (96) | (67) | (15.3) |
| 1965 | | 121[c] | -- | 12.0[d] | 114.0[d] | 8.0[d] | (34) | (44) | (8.8) | (97) | (66) | (15.4) |
| 1964 | | | 6.8[d] | 17.0[d] | 127.0[d] | 7.3[d] | (35) | (34) | (8.9) | (100) | (51) | (15.7) |
| 1963 | | | | (22) | 122.0[d] | 8.5[d] | (29) | (34) | (8.3) | (83) | (51) | (14.1) |
| 1962 | | | | 26.0[d] | 151.0[d] | (11.6) | (39) | (37) | (9.3) | (112) | (55) | (16.7) |
| 1961 | | | | (27) | 145.0[d] | (11.0) | (37) | (36) | (9.1) | (106) | (54) | (16.2) |
| 1960 | | | | (22) | 121.0[d] | 7.7[d] | (28) | (37) | (8.2) | (79) | (55) | (13.8) |
| 1959 | | | | (22) | 117.0[d] | 6.5[d] | (25) | (24) | (7.9) | (72) | (37) | (13.1) |
| 1958 | | | | (19) | 101.0[d] | (6.6) | (23) | (32) | (7.7) | (65) | (48) | (12.5) |
| 1957 | | | | (24) | 132.0[d] | (9.7) | (28) | (40) | (8.2) | (80) | (60) | (13.9) |
| 1956 | | | | (30) | 163.0[d] | (12.8) | (50) | (42) | (10.4) | (143) | (64) | (19.5) |
| 1955 | | | | (36) | 195.0[d] | (16.1) | (50) | (39) | (10.4) | (142) | (59) | (19.4) |
| 1954 | | | | (36) | 195.0[d] | (16.2) | (55) | (36) | (10.9) | (157) | (54) | (20.8) |
| 1953 | | | | (28) | 153.0[d] | (11.8) | (63) | (46) | (11.7) | (178) | (68) | (22.7) |
| 1952 | | | | | | | | | | (186) | (72) | (23.4) |
| 1951 | | | | | | | | | | (192) | (69) | (23.9) |
| 1950 | | | | | | | | | | (208) | (71) | (25.4) |
| 1949 | | | | | | | | | | (177) | (50) | (22.6) |
| 1948 | | | | | | | | | | (197) | (57) | (24.4) |
| 1947 | | | | | | | | | | (197) | (68) | (24.4) |
| 1946 | | | | | | | | | | (152) | (29) | (20.3) |
| 1945 | | | | | | | | | | (184) | (57) | (23.2) |
| 1944 | | | | | | | | | | (235) | (77) | (27.8) |
| 1943 | | | | | | | | | | (284) | (83) | (32.2) |
| 1942 | | | | | | | | | | (286) | (81) | (32.4) |
| 1941 | | | | | | | | | | (313) | (69) | (34.8) |
| 1940 | | | | | | | | | | (310) | (63) | (34.6) |

[a] Pollutants are identified as follows: $SO_2$ - sulfur dioxide, TSP - total suspended particulate, SS - suspended sulfate. All values are annual arithmetic means. Estimated values are in parentheses.

For Kearns and Magna, estimates of annual $SO_2$ exposures were derived by multiplying the yearly smelter emission of $SO_2$ by the ratio of the 1971 measured annual average $SO_2$ concentration to the 1971 $SO_2$ emission rate (193 tons/day).

For Kearns and Magna, estimates of suspended sulfates were derived from estimates of $SO_2$, using the following regression equations for 1971:

$$\text{Kearns - SS} = 0.10\ (SO_2) + 5.41$$
$$\text{Magna - SS} = 0.09\ (SO_2) + 6.66$$

For Kearns and Magna, estimates of annual TSP exposures were derived by multiplying the yearly smelter production of copper by the ratio of the 1971 measured annual arithmetic mean TSP concentration to the 1971 copper production rate (260,000 tons/year). The assumption is made that the smelter is the main source of TSP in Kearns and Magna. For Salt Lake City, estimates of suspended sulfates were derived from annual average concentrations of TSP using the following regression equation:

$$\text{Salt Lake City - SS} = 0.101\ (\text{TSP}) - 3.65$$

This equation was derived using all annual data from Salt Lake City in which both SS and TSP levels were known. The correlation coefficient is 0.74. $SO_2$ concentrations for Salt Lake City were derived using an average ratio of $SO_2$ to TSP for all available Salt Lake City data.

[b] CHESS data.

[c] Utah State Division of Health data.

[d] National Air Surveillance Network data.

# 2.2 PREVALENCE OF CHRONIC RESPIRATORY DISEASE SYMPTOMS IN ADULTS: 1970 SURVEY OF SALT LAKE BASIN COMMUNITIES

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## INTRODUCTION

Air pollution episodes characterized by extremely high levels of sulfur dioxide and total suspended particulates are associated with increased respiratory disease symptoms and increased mortality.[1-4] Studies of the chronic effects of relatively low levels of sulfur dioxide have been less conclusive. Bell, Petrilli et al., and Tsunetoshi et al. found positive associations between low-level sulfur dioxide concentrations and chronic bronchitis.[5-7] Holland et al.,and Holland and Reid likewise found an increased number of episodes of cough, phlegm, and chest illnesses in men who lived in a polluted area of London. British males age 50 to 59 living in London were compared to men of the same ages in three towns where the sulfur dioxide concentrations were lower and found to manifest more frequent and more severe chronic respiratory disease symptoms.[8,9] In contrast, Zeidberg et al. could find no relationship between respiratory diseases and very low-level sulfur dioxide concentrations.[10] Furthermore, Anderson, Ferris, and Zichmantel found no association between the prevalence of chronic respiratory disease and low levels of sulfur dioxide.[11-13] Most of these studies were hampered by an inability to separate the effects of sulfur dioxide from those of suspended particulates. Inadequate air monitoring and intervening variables such as age, socioeconomic status, and smoking habit limit the scope of most earlier studies.

The purpose of this paper is to describe a survey designed to estimate the effect of sulfur dioxide and suspended sulfate exposure on the prevalence of chronic respiratory disease symptoms. The study was conducted in communities that have similar total suspended particulate levels but represent an exposure gradient for sulfur dioxide and suspended sulfates, which largely emanate from a single industrial source, a copper smelter. The corporation involved began reducing the emissions of air pollutants several years ago and is making further substantial efforts to control pollutant discharges.

Four specific hypotheses were tested: first, that exposure to elevated community air pollution levels in the immediate vicinity of the industrial source would be accompanied by significantly increased indices of chronic respiratory disease frequency or severity; second, that the prevalence of chronic respiratory disease symptoms in the more polluted community would increase with increasing length of residence when compared to populations living in cleaner communities; third, that the effects of community air pollution and self-pollution by smoking cigarettes would be additive; and fourth, that relatively brief peak exposures in a community somewhat more distant from the point exposure source would be accompanied by increases in one or more indices of chronic respiratory disease morbidity.

## MATERIALS AND METHODS

### Community Selection

On the basis of existing aerometric, meteorologic, and topographic data, four Salt Lake Basin communities were chosen to represent a gradient of sulfur dioxide exposure. These four communities were: a section of Ogden (the Low exposure area), a section of Salt Lake City (the Intermediate I exposure area), Kearns (the Intermediate II exposure area), and Magna (the High exposure area). The primary source of sulfur dioxide emissions was a large copper smelter located approximately 5 miles northwest of the High exposure community. The Intermediate II exposure community is located approximately 8 miles southeast of the smelter, the Intermediate I exposure neighborhood approximately 13 miles east of the smelter, and the Low exposure community approximately 38 miles northeast of the smelter.

### Collection of Health and Demographic Data

During autumn 1970, chronic respiratory disease information was ascertained from parents of elementary, junior high, and senior high school students. In each of the four communities, three elementary schools, one junior high school, and one senior high school were selected on the basis of similar socioeconomic status. Each community was a middle-class, predominantly white area. Elementary school children carried home the questionnaire, while parents of junior and senior high school students were contacted by mail.

The questionnaire, which was adapted for self-administration from that version standardized by the British Medical Research Council, inquired about the duration and presence of cough, phlegm (excluding phlegm from the nose), and shortness of breath. The reliability of this type of self-administered questionnaire has been tested and found to be very satisfactory.[7] An example of the form used is presented elsewhere.[14]

The mother or female guardian was instructed to answer the questions for parents living in the home. Other information ascertained included length of residence in the community, educational attainment of head of household, cigarette smoking habit, age, and whether the parent had occupational exposure to respiratory irritants. It should be made clear that covariate information was applicable to the time at which the survey was conducted. For example, exsmokers were individuals who at the time of the survey were not smoking, but who had smoked in the past.

## Assessing Air Pollution Exposure

At the time of this study, air monitoring stations were located within each community. Each station was on a building rooftop at a height of about 25 feet above the ground. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high-volume samplers), suspended sulfates, and suspended nitrates were monitored daily. Daily measurements of nitrogen dioxide (Jacobs-Hochheiser method) were also made; however, the validity of the method has subsequently been questioned by the Environmental Protection Agency. Dustfall was determined from monthly samples. A full description of the monitoring station locations and aerometric procedures has been presented elsewhere.[15]

While these stations allow estimates of pollutant exposure for prospective studies, it was also necessary to estimate exposure for past time periods. Sulfur dioxide had been monitored in the Low and Intermediate I communities since 1965 and in the High community since 1970 by the Utah State Division of Health. In addition, measurements of total suspended particulates and suspended sulfates dating back to 1953 were available for the Intermediate I community. Estimates of sulfur dioxide, total suspended particulates, and suspended sulfate concentrations in the High exposure community for 1940-1970 and the Intermediate II exposure community for 1950-1970 were obtained by a mathematical dispersion model, which utilized emissions from the industrial source and extensive local meteorological data, and by observed relationships among pollutants. Observed suspended particulate, suspended sulfate, and sulfur dioxide concentrations for 1970-1971 were used to calibrate the models used to estimate exposure levels for previous years.

## Data Analysis

For the purposes of statistical analysis, a scale of symptom severity was devised by a panel of physicians based upon the number, clinical significance, and duration or persistence of symptoms. Although the number of symptom categories was somewhat arbitrary and their severity ranking was subjective and no more than ordinal in nature, the following ranking of chronic respiratory symptoms categories was developed:

1. No symptoms.
2. Cough alone for less than 3 months each year.
3. Phlegm with or without cough for less than 3 months each year.
4. Cough without phlegm for 3 months or more each year.
5. Phlegm without cough for 3 months or more each year.
6. Cough and phlegm for 3 months or more each year.
7. Cough and phlegm for 3 months or more each year and shortness of breath.

Reported respiratory symptoms were used to devise three indices of morbidity; first, chronic bronchitis was defined in accordance with the British Medical Research Council (cough and phlegm on most days for at least 3 months each year), and prevalence rates were compared across areas. The second variable was a mean respiratory symptom score defined as the arithmetic average of the respiratory symptom score of each member of a particular population group. The third variable was the arithmetic average of the respiratory symptom score for those members of the population who reported any chronic respiratory symptoms, that is, for those members who had symptom scores 2 through 7 only. The first two variables reflect frequency of chronic respiratory disease symptoms, with the mean symptom score having the advantage of considering a gradient of symptoms but the disadvantage of lacking the acceptance and the assured clinical relevance of classically defined chronic bronchitis. The mean severity score, which is the third variable, was utilized as a measure of severity of reported symptoms.

Hypotheses were tested using a general linear model for categorical data.[16] This technique uses weighted regression on categorical data and allows estimation of each effect in the model after adjusting for all other effects in the model. Hypotheses were tested by Chi Square procedures. Covariates used in the analysis model were age and cigarette smoking habit. Interaction terms were not included.

## RESULTS

### Environmental Exposure

Pollutant exposures already projected from aerometric and dispersion model estimates were averaged for each community by decade from 1940 through 1959 and by 4-year intervals thereafter (Table 2.2.1). More extensive aerometric data for the year 1971 are isolated in the table for comparative purposes. Procedures utilized in these projections are detailed elsewhere.[15]

Estimated arithmetic mean sulfur dioxide levels for the period 1950-1970 show that the community exposure gradient very likely existed for two decades prior to this study. During this two-decade period, estimated annual average sulfur dioxide levels in all communities, except the town nearest the industrial source, have been quite low (17 to 50 $\mu g/m^3$) and well within the National Annual Primary Ambient Air Quality Standard for sulfur oxides. In the High exposure community, estimated annual levels of sulfur dioxide have been falling steadily from a high of 234 $\mu g/m^3$ three decades ago to 92 to 95 $\mu g/m^3$ for 1960-1970. For 1971, the levels were somewhat lower, 62 $\mu g/m^3$.

Two factors accounted for lower sulfur dioxide levels during 1971 in the High exposure community. New emission controls for the industrial source were implemented in 1971. In addition, a near cessation of industrial activity in July 1971 caused an extreme drop in ambient levels of sulfur dioxide.

In the 3-year period before the survey was conducted, 1968-1970, total suspended particulate levels were minimally elevated (84 to 88 $\mu g/m^3$) in the Low and Intermediate I exposure areas. Earlier annual exposures (1950-1967) in the Intermediate I community were probably moderately high (103 to 151 $\mu g/m^3$). In the other two communities, sus-

Table 2.2.1. AIR POLLUTANT EXPOSURE ESTIMATES ($\mu g/m^3$) IN FOUR UTAH COMMUNITIES, 1940-1971

| Pollutant and community | 1940-49 | 1950-59 | 1960-63 | 1964-67 | 1968-70 | 1971 |
|---|---|---|---|---|---|---|
| **Sulfur dioxide** | | | | | | |
| Low | NA[a] | NA | NA | NA | NA | 8 |
| Intermediate I | NA | 28 | 27 | 17 | 18 | 15 |
| Intermediate II | —[b] | 50 | 33 | 33 | 32 | 22 |
| High | 234 | 142 | 95 | 95 | 92 | 62 |
| **Total suspended particulates** | | | | | | |
| Low | NA | NA | NA | 108 | 88 | 78 |
| Intermediate I | NA | 151 | 134 | 103 | 84 | 81 |
| Intermediate II | —[b] | <50 | <50 | <50 | <50 | 45 |
| High | 63 | 60 | 53 | 57 | 70 | 66 |
| **Suspended sulfate** | | | | | | |
| Low | NA | NA | NA | 5.8 | 3.7 | 5.6 |
| Intermediate I | NA | 11 | 8 | 7.7 | 4.7 | 7.3 |
| Intermediate II | —[b] | 10 | 8.7 | 8.7 | 8.6 | 7.8 |
| High | 28 | 20 | 15.2 | 15.3 | 15.0 | 12.4 |
| **Suspended nitrates[c]** | | | | | | |
| Low | — | — | — | — | — | 2.7 |
| Intermediate I | — | — | — | — | — | 3.3 |
| Intermediate II | — | — | — | — | — | 2.4 |
| High | — | — | — | — | — | 2.0 |

[a] NA—not available.
[b] The Intermediate II city did not exist until 1953.
[c] Only fragmentary data available for suspended nitrates from 1940-1970.

pended particulate levels (<50 to 70 $\mu g/m^3$) were thought to have been well below the National Annual Primary Ambient Air Quality Standard for the last three decades. Estimated and measured suspended particulate sulfates evidenced a community gradient in pollution exposure similar to that for sulfur dioxide, being exceedingly low (<5.8 $\mu g/m^3$) in the Low exposure community, moderate (5 to 11 $\mu g/m^3$) in the Intermediate I and II communities, and high (12 to 28 $\mu g/m^3$) near the industrial emissions source. Suspended particulate nitrate levels (2.0 to 3.3 $\mu g/m^3$) were similar for all areas.

Three air quality trends should be highlighted: First, there appeared to be a general decrease in the levels of sulfur dioxide and particulate air pollution over the last decade, which was probably due, at least in part, to the pollution control efforts of local industry and governmental units. Second, the Intermediate I and II communities, as well as the High community, appeared to experience elevated annual suspended sulfate levels attributable to the point source of industrial emissions. Annual average sulfur dioxide levels in the Intermediate I and II communities were not greatly elevated. Third, there appeared to be similar community exposure gradients for sulfur dioxide and suspended sulfates, which makes it difficult to separate the effects of these pollutants.

## Community Characteristics

Four community characteristics of special importance in the assessment of chronic respiratory disease are respondent rates, exclusion rates, socioeconomic status, and cigarette smoking habits. Parents of elementary school children completed and returned 87 percent of the questionnaires, while parents of junior and senior high children, to whom questionnaires were mailed, completed and returned 35 percent of the questionnaires (Table 2.2.2). For both groups of parents, intercommunity differences were observed. Parents of elementary school children in the two Intermediate exposure areas returned a somewhat smaller proportion of questionnaires than the High and Low exposure communities ($p < 0.001$). Parents of junior and senior high school children in the Low and Intermediate I exposure areas returned smaller proportions of questionnaires than in the Intermediate II and High exposure communities ($p < 0.001$).

Because 2 years seemed a minimum time in which chronic respiratory disease might develop, respondents were excluded from the analyses when they had not lived in their community for at least 2 years prior to the survey. Parents were also excluded if they reported occupational exposures to irritating fumes, dusts, or aerosols. Lastly, parents were excluded if information needed for the analyses was missing from the questionnaire. The percent of mothers excluded was approximately the same in all four communities; more fathers were excluded in the High exposure community than in other communities because many fathers from the High exposure community worked in a nearby industry where they reported occupational exposure to irritants, fumes, dusts, or gases, including sulfur dioxide (Table 2.2.2). A sample of 27 to 36 nonrespondent families from each community were interviewed by telephone and found to be similar to respondents in education, smoking habits, family size, and length of residence.

Table 2.2.2. COMMUNITY CHARACTERISTICS: QUESTIONNAIRE RESPONSE, EXCLUSIONS, AND EDUCATIONAL ATTAINMENT OF FATHERS

| Community | Percent of families responding | | Percent of respondents excluded[a] | | Percent of fathers who completed high school |
|---|---|---|---|---|---|
| | Distributed through elementary school | Mailed to families of junior and senior high school students | Mothers | Fathers | |
| Low | 91 | 34 | 16 | 27 | 78 |
| Intermediate I | 83 | 32 | 21 | 34 | 66 |
| Intermediate II | 86 | 40 | 19 | 35 | 68 |
| High | 88 | 37 | 22 | 50 | 73 |
| Total | 87 | 35 | 19 | 37 | 71 |

[a] Because of incomplete response, less than 2 years residence, or occupational exposure.

Among the families responding to the questionnaire, there were small intercommunity differences in educational achievement (Table 2.2.2). The Low exposure area had an educational ranking only slightly higher than the High exposure community, which, in turn, ranked above the two Intermediate exposure areas. This intercommunity socioeconomic pattern could hardly diminish intercommunity differences in chronic respiratory disease that might be attributable to residence in the High exposure community because comparison communities ranked socioeconomically higher and lower than the High exposure community.

Sex and cigarette smoking are such strong determinants of chronic respiratory disease that the study population of each community was categorized by sex and history of cigarette smoking before chronic respiratory disease morbidity indices were evaluated (Table 2.2.3). There were no significant intercommunity differences in the intensity of smoking among respondents who currently smoked cigarettes. Likewise, cigarette smokers of both sexes living in the High exposure community were no more likely to be heavy smokers (>1-1/2 packs per day) than their counterparts in the other three communities. When the smoking intensity of either mothers or fathers was considered, the High exposure community ranked midway among the four study areas. The low percentage (28 percent) of parents who reported being current smokers can be explained by the fact that the study communities were composed largely of individuals of a religious group whose members are typically nonsmokers.

## Chronic Bronchitis Prevalence

The suspected determinants of chronic bronchitis other than ambient air pollution were first evaluated by calculation of sex-specific chronic bronchitis prevalance rates (Table 2.2.4). As expected, cigarette smoking proved the strongest determinant, followed by sex and educational attainment. However, educational attainment was linked to cigarette smoking, with those who were less educated more likely to be cigarette smokers. Within sex- and smoking-specific categories, no consistent trend that was attributable to educational attainment could be found. For all but one of the examined categories—smoking, education, and age—fathers were found to have higher rates than mothers. The only exception to this sex effect was a somewhat lower chronic bronchitis prevalence among fathers who had stopped smoking cigarettes. Little or no differences could be attributed to aging in this population, probably because of the narrow age span. Seemingly elevated rates in the 29-year-or-younger group were based upon a relatively small sample size.

To determine the effects of ambient air pollution on the prevalence of chronic bronchitis, smoking- and sex-specific rates for each community were calculated, and these revealed a clear trend towards excess illness in the High exposure community (Table 2.2.5). In four of six possible smoking- and sex-specific comparisons, chronic bronchitis rates in the Intermediate II exposure community fell below the markedly elevated rates of the High exposure area and above the relatively low rates of the Low and

Table 2.2.3. STUDY POPULATION DISTRIBUTED BY COMMUNITY, SEX, AND SMOKING STATUS

| Community | Nonsmokers | | Exsmokers | | Smokers | | Total |
|---|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers | |
| Low | 755 | 396 | 75 | 230 | 214 | 272 | 1942 |
| Intermediate I | 755 | 367 | 101 | 177 | 286 | 311 | 1997 |
| Intermediate II | 772 | 350 | 114 | 241 | 295 | 354 | 2126 |
| High | 667 | 265 | 84 | 133 | 212 | 209 | 1570 |
| Total | 2949 | 1378 | 374 | 781 | 1007 | 1146 | 7635 |

**Table 2.2.4. SEX-SPECIFIC CHRONIC BRONCHITIS PREVALENCE DISTRIBUTED BY SMOKING HISTORY, EDUCATIONAL ATTAINMENT, AND AGE**

| Determinant | Chronic bronchitis prevalence, percent | | |
|---|---|---|---|
| | Mothers | Fathers | Both sexes |
| **Smoking history:** | | | |
| Nonsmokers | 3.5 | 3.7 | 3.6 |
| Exsmokers | 5.9 | 4.2 | 4.8 |
| Smokers | 17.1 | 20.9 | 18.3 |
| **Education:** | | | |
| <High school | 7.7 | 13.8 | 10.3 |
| High school | 7.2 | 9.1 | 8.0 |
| >High school | 6.1 | 8.1 | 7.0 |
| **Age:** | | | |
| ≤29 | 9.4 | 13.4 | 10.5 |
| 30 to 39 | 6.2 | 9.6 | 7.6 |
| 40 to 49 | 7.1 | 8.8 | 7.9 |
| ≥50 | 4.2 | 10.5 | 8.1 |

**Table 2.2.5. SMOKING- AND SEX-SPECIFIC CHRONIC BRONCHITIS PREVALANCE RATES (percent) DISTRIBUTED BY COMMUNITY**

| Community | Nonsmokers | | Exsmokers | | Smokers | |
|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers |
| Low | 2.3 | 3.0 | 5.3 | 2.6 | 17.8 | 19.9 |
| Intermediate I | 2.0 | 3.6 | 4.0 | 3.4 | 14.7 | 18.6 |
| Intermediate II | 4.7 | 2.3 | 7.0 | 5.4 | 15.3 | 20.1 |
| High | 5.2 | 6.8 | 7.1 | 6.0 | 22.2 | 26.8 |

Intermediate I exposure communities. Rates were highest for current cigarette smokers of either sex who lived in the most polluted community.

In five of the eight possible intracommunity comparisons between exsmokers and nonsmokers in Table 2.2.5, chronic bronchitis rates for exsmokers were somewhat higher than those of lifetime nonsmokers. In every intracommunity comparison, rates for exsmokers were distinctly lower than those for current cigarette smokers. Differences between the sexes were inconsistent across smoking categories. Among current cigarette smokers, males consistently exhibited a higher chronic bronchitis prevalence, while the converse was true for exsmokers. No real sex pattern could be found among nonsmokers.

Tests for statistical significance of these effects, as well as the effect of age, are presented separately for mothers and fathers in Table 2.2.6. Significant increases in the prevalence of chronic bronchitis in both sexes were associated with cigarette smoking and residence in the more polluted community. Significant increases did not occur in the Intermediate II exposure area. The nonsignificant Chi Squares for the fit of the models indicate that linear models containing only smoking, age, and community pollution exposure effects adequately fit the data.

An additional analysis was performed with the data on the parents of junior and senior high school students omitted. In this analysis, the excess in the prevalence of chronic bronchitis in the High exposure

Table 2.2.6. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS, CHRONIC BRONCHITIS RATES

| Determinant | Degrees of freedom | Fathers | | Mothers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability | $X^2$ | Probability |
| Pollutant exposure | 3 | 11.15 | $p<0.001$ | 21.26 | $p<0.001$ |
| Smoking[a] | 1 | 178.23 | $p<0.001$ | 116.72 | $p<0.001$ |
| Age | 1 | 3.12 | $0.05<p<0.1$ | 2.01 | $0.1<p<0.2$ |
| Fit of model | 10 | 8.54 | $0.5<p<0.6$ | 10.37 | $0.4<p<0.5$ |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a better sample size.

community was larger than when these parents were included in the data.

## Mean Symptom and Severity Scores for Respiratory Symptoms

Smoking- and sex-specific respiratory symptom and severity scores were also summarized for the four communities. Overall symptom scores were first averaged for the total population (Table 2.2.7). The mean respiratory symptom score for residents of the most polluted community, like the chronic bronchitis prevalence rate, was consistently higher than rates for the Low and Intermediate exposure communities. The symptom scores followed the pollution gradient for sulfur dioxide and suspended particulate sulfates in all but two of the smoking- and sex-specific intercommunity comparisons. In both of these cases, the score of the High exposure community was still much higher than the score of any one of the other three communities. Statistical tests involving overall mean symptom scores duplicated those previously described for chronic bronchitis, with smoking and ambient air pollution both being significant determinants of morbidity (Table 2.2.8). Thus, the mean symptom score analysis both confirmed the analysis for chronic bronchitis rates and suggested that more subtle increments in respiratory illness might be occurring in the Intermediate II exposure community.

Smoking- and sex-specific severity scores for that portion of the population who reported respiratory symptoms were then calculated (Table 2.2.9) and statistically tested by the previously described linear model (Table 2.2.10). Intercommunity differences in this conditional severity score followed no clear pattern. However, age-specific severity scores, which were calculated but are not included, showed unmistakable evidence of increasing severity in current cigarette smokers and with increasing age. This was confirmed by statistical testing. Together, the two score analyses demonstrated that the frequency of chronic respiratory illness, but not the severity of such illnesses, was increased in the most polluted community.

Table 2.2.7. SMOKING- AND SEX-SPECIFIC MEAN RESPIRATORY SYMPTOM SCORES DISTRIBUTED BY COMMUNITY

| Community | Nonsmokers | | Exsmokers | | Smokers | |
|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers |
| Low | 1.36 | 1.54 | 1.65 | 1.48 | 2.57 | 2.73 |
| Intermediate I | 1.37 | 1.56 | 1.79 | 1.54 | 2.53 | 2.88 |
| Intermediate II | 1.55 | 1.48 | 1.88 | 1.82 | 2.44 | 2.99 |
| High | 1.73 | 1.94 | 2.02 | 1.87 | 2.98 | 3.32 |

**Table 2.2.8. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS, MEAN RESPIRATORY SYMPTOM SCORES**

| Determinant | Degrees of freedom | Fathers | | Mothers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability | $X^2$ | Probability |
| Pollutant exposure | 3 | 25.62 | $p<0.001$ | 38.71 | $p<0.001$ |
| Smoking[a] | 1 | 344.58 | $p<0.001$ | 221.82 | $p<0.001$ |
| Age | 1 | 1.75 | $0.1<p<0.2$ | 0.11 | $0.7<p<0.8$ |
| Fit of model | 10 | 10.67 | $0.3<p<0.4$ | 9.74 | $0.4<p<0.5$ |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a better sample size.

**Table 2.2.9. SMOKING- AND SEX-SPECIFIC MEAN RESPIRATORY SEVERITY SCORES IN THE PRESENCE OF ANY CHRONIC RESPIRATORY DISEASE SYMPTOM DISTRIBUTED BY COMMUNITY**

| Community | Nonsmokers | | Exsmokers | | Smokers | |
|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers |
| Low | 4.19 | 4.55 | 4.50 | 4.47 | 4.85 | 4.88 |
| Intermediate I | 4.05 | 4.36 | 4.33 | 4.10 | 4.38 | 4.58 |
| Intermediate II | 4.71 | 4.27 | 4.70 | 4.28 | 4.52 | 4.68 |
| High | 4.30 | 4.47 | 4.19 | 4.22 | 4.74 | 4.79 |

**Table 2.2.10 ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS, MEAN RESPIRATORY SEVERITY SCORES**

| Determinant | Degrees of freedom | Fathers | | Mothers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability | $X^2$ | Probability |
| Pollutant exposure | 3 | 5.49 | $0.1<p<0.2$ | 11.87 | $p<0.01$ |
| Smoking[a] | 1 | 19.96 | $p<0.001$ | 8.03 | $p<0.01$ |
| Age | 1 | 10.34 | $p<0.01$ | 7.11 | $p<0.01$ |
| Fit of model | 10 | 4.35 | $p>0.9$ | 11.30 | $0.3<p<0.4$ |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a better sample size.

## Occupational Exposure

Fathers in the High exposure community with confirmed occupational exposure to irritant dusts, fumes, gases, or aerosols were compared to the fathers who were not occupationally exposed to pollutants. There were 174 nonsmoking and 105 smoking occupationally exposed fathers, and 385 nonsmoking and 205 smoking fathers who were not occupationally exposed. Preliminary analyses indicated that age was interacting with exposure, hence separate analyses were done on the age groups over and under 40. For the age group under 40, there was no significant difference between occupationally exposed and unexposed fathers in either chronic bronchitis prevalence, symptom scores, or severity scores (Table 2.2.11). Significantly higher chronic bronchitis prevalence rates and mean symptom scores were seen in a similar comparison involving older occupationally exposed fathers. Conditional severity scores were also increased, but not significantly, in older exposed workers.

The net effect of excluding occupationally exposed parents from the main analyses was to diminish any effects attributable to pollution. The incremental effect of occupational exposure in the older workers was roughly twice the effect attributable to ambient air pollution and over half that of cigarette smoking. Occupational exposures to air pollutants, self-pollution by cigarette smoking, and ambient air pollution appear to be additive hazards. Safety standards set to protect workers from inhaled irritants will be imprecise, if not ineffective, unless self-pollution by smoking and ambient air pollution exposures are duely considered in the standard-setting process.

## Length of Residence

Two questions prompted analysis of duration of residence in the study communities: first, did community air pollution influence migration patterns by deterring more vulnerable citizens from moving into a polluted community or by increasing out-migration of citizens whose ill health may be aggravated by pollution? Second, what length of exposure to the pollutant levels found in the present study was associated with significant increases in chronic bronchitis prevalence?

Relative prevalence ratios and expected chronic bronchitis prevalence rates were computed for five residence categories in the High exposure community based upon the pooled experience of the three Low and Intermediate exposure communities. Smoking-specific relative prevalence plots suggested that a deficit of persons with chronic bronchitis moved into the High exposure area and a probable excess moved out after a lapse of 10 to 12 years (Figure 2.2.1). This effect was most pronounced in the current nonsmokers group, which was composed of exsmokers and lifetime nonsmokers. Possibly, people subject to chronic bronchitis avoided self-pollution by not smoking and minimized ambient air pollution by their choice of occupation and residence. Comparison of the observed and expected chronic bronchitis rates at successively greater lengths of residence established that significant increases in illness occurred both in smokers and current nonsmokers after 4 to 7 years.

## Relative Effect of Community Air Pollution and Cigarette Smoking

Cigarette smoking and ambient air pollution are both significant determinants of chronic bronchitis

**Table 2.2.11. SMOKING - ADJUSTED CHRONIC BRONCHITIS RATES, MEAN RESPIRATORY SYMPTOM SCORES, AND MEAN RESPIRATORY SEVERITY SCORES BY OCCUPATIONAL EXPOSURE IN THE HIGH EXPOSURE COMMUNITY**

| Occupational exposure to smoke, dust, or fumes | Chronic bronchitis rate, percent | | Overall mean severity scores | | Mean severity scores of fathers with symptoms | |
|---|---|---|---|---|---|---|
| | <40 yr olds | ≥40 yr olds | <40 yr olds | ≥40 yr olds | <40 yr olds | ≥40 yr olds |
| Yes | 18.4 | 25.9[a] | 2.56 | 3.26[a] | 4.63 | 5.07 |
| No | 16.0 | 17.9 | 2.60 | 2.58 | 4.54 | 4.67 |

[a] Significantly increased.

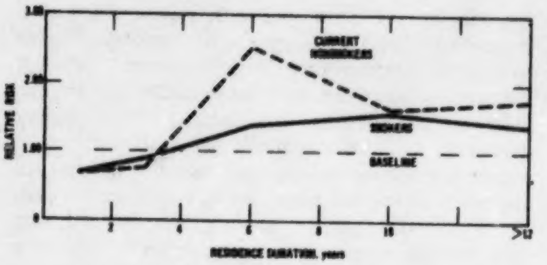

Figure 2.2.1. Relative risk of chronic bronchitis in High exposure community compared to pooled risk for all other communities.

morbidity, but what is the relative importance of these insults? Are their effects additive? To answer these questions, the experience of lifetime nonsmokers living in the three relatively clean communities was used to compute a base prevalence rate for each sex separately. For each smoking and community pollution category, the excess prevalence was calculated by subtracting the appropriate base rate from the smoking and community specific prevalence rate (Table 2.2.12).

Should the deleterious effects of cigarette smoking and ambient air pollution be additive, then the sum of the excess prevalence for smokers living in clean communities plus the excess prevalence for nonsmokers from the polluted community should equal the excess prevalence for smokers living in the polluted community. When these excess prevalences were summed, both sexes had somewhat higher excess prevalences (117 and 128 percent) than predicted by the additive model. When the same additive model was applied to exsmokers, air pollution and cigarette smoking again appeared to be additive (64 and 79 percent) of the predicted value. To assess the relative importance of air pollution where the effects of air pollution and cigarette smoking are additive, the excess prevalence of chronic bronchitis for nonsmokers in the most polluted community may be divided by the excess prevalence for current cigarette smokers living in cleaner communities. When this was done for males, air pollution

Table 2.2.12. RELATIVE IMPORTANCE OF CIGARETTE SMOKING AND AMBIENT AIR QUALITY AS DETERMINED BY COMPARISON OF EXCESS PREVALENCE FOR CHRONIC BRONCHITIS

| Current smoking status | Community air quality | Excess prevalence of chronic bronchitis[a] | Simple additive model combining adverse effects of smoking and pollution | |
|---|---|---|---|---|
| | | | Expected | Observed/ expected |
| **Females** | | | | |
| Lifetime nonsmokers | Clean (pooled)[b] | 0.00 (3.0) | – | – |
| | Dirty[c] | 2.2 | – | – |
| Exsmokers | Clean (pooled) | 3.0 | – | – |
| | Dirty | 4.1 | 5.2 | 0.79 |
| Smokers | Clean (pooled) | 12.8 | – | – |
| | Dirty | 19.2 | 15.0 | 1.28 |
| **Males** | | | | |
| Lifetime nonsmokers | Clean (pooled) | 0.00 (3.0) | – | – |
| | Dirty | 3.8 | – | – |
| Exsmokers | Clean (pooled) | 0.9 | – | – |
| | Dirty | 3.0 | 4.7 | 0.64 |
| Smokers | Clean (pooled) | 16.6 | – | – |
| | Dirty | 23.8 | 20.4 | 1.17 |

[a] Base rates in parentheses. Excess prevalence = smoking- and community-specific prevalence rate minus base rate.
[b] Based on the Low, Intermediate I, and Intermediate II exposure communities combined.
[c] Based on the High exposure community.

was found to have about one-fourth (0.23) the effect of cigarette smoking; for females, air pollution was found to have about one-tenth (0.09) the effect of cigarette smoking. In summary, excess prevalence comparisons indicate that a large portion of excess chronic bronchitis morbidity can clearly be attributed to ambient air pollution and that the combined effects of pollution and cigarette smoking appear to be additive.

## DISCUSSION

Three of the four hypotheses advanced in this study were validated by the analyses. The frequency of chronic bronchitis and overall mean respiratory symptom scores were significantly increased among current nonsmokers and cigarette smokers of both sexes who lived in the High exposure community. There was, however, no evidence that the severity of chronic respiratory disease symptoms differed between communities. Chronic bronchitis prevalence was found to increase significantly after 4 to 7 years exposure to the elevated ambient air pollution levels found in the most polluted community. Excess prevalence comparisons indicated that excess chronic bronchitis morbidity attributed to community air pollution and cigarette smoking are additive. This study could not differentiate the effects of elevated annual average sulfur dioxide concentrations from those of peak exposures to sulfur dioxide. Furthermore, where sulfur dioxide levels were elevated, suspended sulfates were also quite high. However, in the Intermediate I and II communities, exposures to elevated levels of suspended sulfates occurred in the absence of elevated annual average exposures of sulfur dioxide. These communities did not differ significantly from the cleaner community, even though there was a trend towards more chronic bronchitis and higher mean symptom scores for respiratory symptoms. One might interpret this finding as suggesting that annual average exposures to suspended sulfates of 11 to 12 $\mu g/m^3$ could be near the threshold for induction or magnification of chronic respiratory disease symptoms. Since past suspended particulate exposures in the Low and Intermediate I exposure areas had been moderately elevated, it is possible that these less polluted communities may still harbor a small residual morbidity excess, making the present estimates of air pollution effects conservative.

There was also some evidence that people with chronic respiratory disease symptoms hesitated to migrate to the most polluted community and a suggestion that residents who developed such symptoms preferentially migrated from the High exposure community to a more salubrious climate. Ambient air pollution was found to have roughly one-tenth to one-fourth the deleterious effect of cigarette smoking and one-half that of occupational exposure to irritant gases, dusts, fumes, or aerosols. As expected, cigarette smoking history, sex, and occupational exposures were significant determinants of chronic respiratory disease frequency. Educational attainment and age were not strong determinants in the present study. Moreover, intercommunity differences in educational attainment were small. Each of these variables was duely considered in the analysis.

One intervening variable, the influence of nonrespondents, must be considered further. To equalize the prevalence of chronic bronchitis, major intercommunity differences in morbidity would have to affect nonrespondents in a direction opposite to the differences observed in respondents. To equalize chronic bronchitis rates, nonrespondent smokers in the cleaner areas would have to double illness rates of respondents, and nonrespondent cigarette smokers from the cleaner areas would have to report one-and-one-half more illness than respondents. Such differences are conceivable, but hardly likely. One other exclusion, that of occupationally exposed workers in the High exposure community, clearly diminished intracommunity differences attributable to air pollution.

After the effects of all intervening variables were duly considered, we concluded that it is only prudent from a public health standpoint to assume that increases in the prevalence of chronic bronchitis can be related to 4- to 7- year exposures to ambient air pollution. Such exposures were characterized by elevated levels of sulfur dioxide (92 to 95 $\mu g/m^3$) accompanied by low suspended particulate levels (53 to 70 $\mu g/m^3$) and elevated suspended sulfate levels (15 $\mu g/m^3$) in the absence of elevated levels of suspended nitrates. Clearly, additional clinical and toxicological laboratory research will be necessary to define the relative importance of sulfur dioxide and suspended sulfates. Additional epidemiologic studies will also help piece together the puzzle since urban levels of sulfur dioxide are falling much more rapidly than levels of suspended sulfates. Physical and chemical characterization of suspended sulfates must be improved, and a more complete understanding of the relevant atmospheric chemistry is clearly needed.

## SUMMARY

The prevalence of chronic bronchitis was found to increase significantly among smokers and nonsmokers of both sexes after 4 to 7 years exposure to ambient air pollution characterized by elevated annual levels of sulfur dioxide (92 to 95 $\mu g/m^3$) and suspended sulfates (15 $\mu g/m^3$) accompanied by low levels of total suspended particulates (53 to 70 $\mu g/m^3$). Among the 7635 adults who comprised the study population, chronic bronchitis prevalence was highest in smokers, intermediate in exsmokers, and lowest in lifetime nonsmokers. The effects of air pollution and self-pollution by smoking cigarettes appeared to be additive. Occupational exposure to irritating dusts, fumes, gases, or aerosols was a strong determinant of illness after age 40, having twice the effect of air pollution and over half the effect of cigarette smoking. Community pollution seemed to influence migration patterns in that there was a relative deficit of persons with chronic respiratory disease symptoms among new arrivals in the High exposure community; furthermore, there was evidence that symptomatic persons preferentially migrated away from the High exposure community. All segments of society should gain substantial health benefits with the control of sulfur dioxide air pollution.

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# 2.3 FREQUENCY OF ACUTE LOWER RESPIRATORY DISEASE IN CHILDREN: RETROSPECTIVE SURVEY OF SALT LAKE BASIN COMMUNITIES, 1967-1970

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## INTRODUCTION

Children provide a unique opportunity to assess the effect of air pollutants on the respiratory tract. The young are vulnerable to acute respiratory disease. Unlike adults, children have not usually been subjected to self-pollution by cigarette smoking, to occupational exposures to respiratory irritants, or to a bewildering series of residential changes. Any of these factors can profoundly confound needed estimates of past exposures to ambient air pollutants. When ambient air pollutant levels can be largely attributed to a single industrial point source or when long-term community aerometric data are available, one can determine both recent and past exposures. Furthermore, since air pollution control technology must be directed towards single pollutants, it is especially important to disentangle the effects of multiple pollutant exposures. This has rarely been possible in the past.

British studies of children exposed to particulate and sulfur dioxide pollution indicated a direct relationship between level of exposure and incidence of respiratory disease. Frequency and severity of acute respiratory tract infections increased with the level of exposure to sulfur dioxide and particulates.[1-3] Significant findings have been reported from other countries. School children aged 10 to 11 in a polluted area of Japan had higher frequency of nonproductive cough, irritation of the upper respiratory tract, and increased mucus secretion than matched children in a less polluted area.[4] Similar findings were reported among school children in Irkutsk, U.S.S.R.[5] While these previous studies yielded remarkably consistent results, each suffers from the inability to separate the effects of sulfur dioxide from those of suspended particulates. Moreover, none could relate the length of sulfur dioxide and particulate exposure to excesses in acute respiratory morbidity. However, a single report has linked nitrogen dioxide exposures lasting 3 years to an increase in childhood respiratory illness.[6]

The retrospective survey described here attempted to isolate the effects of sulfur dioxide and suspended sulfate air pollution from the influence of elevated levels of total suspended particulates and oxides of nitrogen. Three specific questions were asked: First, would a greater proportion of children from the polluted communities experience acute lower respiratory illness? Second, would a greater proportion of children from the polluted communities experience repeated episodes of acute lower respiratory illness? Third, would pollutant exposures lasting longer than 2 years alter the susceptibility of children to acute lower respiratory illness?

## MATERIALS AND METHODS

The study was carried out in four communities in the Salt Lake Basin that exhibited similar particulate and nitrogen dioxide pollution levels but represented an exposure gradient for sulfur dioxide and suspended sulfates. These four communities were: an Ogden neighborhood (the Low exposure area), a Salt Lake City neighborhood (the Intermediate I exposure area), Kearns (the Intermediate II exposure area), and Magna (the High exposure area). The primary source of sulfur dioxide emissions was a large smelter located approximately 5 miles northwest of the High exposure community. The Intermediate II exposure community is located approximately 8 miles southeast of the smelter, the Intermediate I exposure neighborhood approximately 13 miles east of the smelter, and the Low exposure community approximately 38 miles northeast of the smelter.

### Collection of Health and Demographic Data

During autumn 1970, lower respiratory illness (LRI) information was ascertained retrospectively by parental reporting for each of their children 12 years old or younger. In each of the four communities, three elementary schools, one junior high school, and one senior high school were selected for study inclusion on the basis of similar socioeconomic status (SES). These areas were each middle-class white communities. Each elementary school child carried home a School and Family Health Questionnaire. Parents of junior and senior high school children were contacted by mail and asked to complete the same questionnaire. (An example of the questionnaire used is presented elsewhere.)[7] Response was encouraged by reminder notes to nonrespondents, and a sample of nonrespondents was interviewed by telephone.

The questionnaire inquired about the frequency of treatment by a physician for pneumonia, croup, or bronchitis (including bronchiolitis or deep chest infections other than pneumonia or croup) during the period beginning in September 1967. Hence all rates used in the analyses (except for the two figures) are 3-year rates. Other information ascertained included hospitalizations for lower respiratory illnesses, name of children's physician, history of asthma, length of residence in the community, socioeconomic status measured by education of head of household, parents' smoking status, and family census. Physician records were reviewed to validate parental reporting for approximately a 15 percent sample of questionnaires. The sample included an equal number of children reported sick and well. Additionally, a sample of 53 physicians in the four areas were queried about their definition of the bronchitis syndrome.

### Assessing Air Pollution Exposure

At the time of this study, air monitoring stations were located within each community. Each station was on a building rooftop at a height of about 25 feet above the ground. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high-volume samplers), suspended sulfates, suspended nitrates, and nitrogen dioxide (Jacobs-Hochheiser method) were monitored daily. Dustfall was determined from monthly samples. A full description of the location of monitoring stations and methodology has been presented elsewhere.[8]

These stations allow estimates of pollutant exposure for prospective studies, but it was also necessary to characterize past exposures. Sulfur dioxide had been monitored in the Low and Intermediate I communities since 1965 and in the High community since 1970 by the Utah State Division of Health. In addition, measurements of total suspended particulates and suspended sulfates dating back to 1953 were available for the Intermediate I community. Estimates of sulfur dioxide, total suspended particulate, and suspended sulfate concentrations in the High exposure community for 1940-1970 and the Intermediate II exposure community for 1953-1970 were obtained by a mathematical dispersion model procedure utilizing emissions output of the industrial source and extensive local meteorological data. Observed suspended particulate, suspended sulfate, and sulfur dioxide concentrations for 1970-1971 were utilized to calibrate the model used to predict exposure levels for previous years.[8]

### Hypotheses Testing

The hypotheses tested were: first, that the frequency of physician treatment for any lower respiratory illness, croup, bronchitis, and pneumonia, as well as the frequency of hospitalization for these illnesses, adjusted for appropriate covariates, would correspond to pollutant exposures; second, that there would be no significant intercommunity differences in the physician definition of lower respiratory illness syndromes for the four communities; and third, that the accuracy of illness reporting by parents would be similar for all communities. For each morbidity condition, two analyses were done. In the first, the dependent variable was the percent of children reporting one or more episodes. For the second, the dependent variable was the percent of children reporting repeated (two or more) episodes.

Hypotheses were tested by a general linear model for categorical data.[9] This technique utilizes weighted regression on categorical data and allows estimation of each effect in the model adjusted for all other effects in the model. Significance was ascertained by Chi Square procedures. Covariates used in the model were age (three categories: 1 to 4, 5 to 8, 9 to 12), sex, and educational attainment (two categories: $<$ completed high school, $\geq$ completed high school), which was an index of socioeconomic status.

## RESULTS

### Environmental Exposure

Pollutant exposures were projected for the illness reporting period covered by the survey (1967-1970) and for the earlier growth and development years of the childhood population (Table 2.3.1). The latter period was partitioned into two 4-year intervals, 1963-1966 and 1959-1962, which reflected changes in air quality and coincided with the earliest exposures of children age 5 through 8 and 9 through 12. Younger children, age 1 through 4, were primarily exposed to pollution levels monitored during the 1967-1970 study period.

The postulated intercommunity exposure gradients were confirmed for sulfur dioxide and suspended sulfates, and such gradients apparently had existed throughout the lifetime of the children surveyed. However, from 1959 to 1970, estimated annual average sulfur dioxide levels were quite low (16 to 33 $\mu g/m^3$) in all but the High exposure community, which apparently had distinct elevations (91 to 94 $\mu g/m^3$). Elevations in estimated suspended sulfates (9 to 15 $\mu g/m^3$) occurred in the two communities near the industrial emissions source. The highest estimated annual sulfate levels were found in the most proximal community.

Annual arithmetic mean suspended particulate levels (82 to 133 $\mu g/m^3$) were moderately elevated in the Low and Intermediate I communities, but not in

Table 2.3.1. PROJECTED AIR POLLUTANT EXPOSURES IN FOUR UTAH COMMUNITIES, 1959-1971

| Pollutant and community | Concentration, μg/m³ | | | |
|---|---|---|---|---|
| | 1959-1962 | 1963-1966 | 1967-1970 | 1971 |
| **Sulfur dioxide** | | | | |
| Low | —[c] | —[c] | —[c] | 8 |
| Intermediate I | 27 | 16 | 18 | 15 |
| Intermediate II | 32 | 33 | 33 | 22 |
| High | 94 | 94 | 91 | 62 |
| **Total suspended particulate** | | | | |
| Low | —[c] | 109 | 92 | 78 |
| Intermediate I | 133 | 114 | 82 | 81 |
| Intermediate II | <50 | <50 | <50 | 45 |
| High | 51 | 59 | 62 | 66 |
| **Suspended sulfates**[a] | | | | |
| Low | —[c] | 6 | 4 | 6 |
| Intermediate I | 8 | 8 | 5 | 7 |
| Intermediate II | 9 | 9 | 9 | 8 |
| High | 15 | 15 | 15 | 12 |
| **Suspended nitrates**[b] | | | | |
| Low | — | — | — | 2.7 |
| Intermediate I | — | — | — | 3.3 |
| Intermediate II | — | — | — | 2.4 |
| High | — | — | — | 2.0 |

[a] Data given for 1959 to 1970 are estimated.
[b] Only fragmentary data are available for suspended nitrates from 1959 to 1970.
[c] Not available.

the Intermediate II and High exposure areas ($<50$ to 62 μg/m³). No data for nitrogen dioxide are given because the validity of the Jacobs-Hochheiser method of analysis has been seriously challenged. However, there was no evidence that the National Primary Ambient Air Quality Standard (100 μg/m³) was exceeded in any of the four communities.

Two factors accounted for relatively lower 1971 annual average of sulfur dioxide in the High exposure community compared with 1959-1970 values. New emission controls for the industrial source were implemented in 1971 and, more importantly, cessation of industrial activity in July 1971 caused a precipitous drop in sulfur dioxide levels.

## Community Characteristics

Intercommunity differences in response rates, nonrespondent characteristics, respondent exclusions, socioeconomic status, family size, and cigarette smoking might alter the interpretation of intercommunity differences in acute respiratory illness reported in the present study. Overall, almost two-thirds of the distributed questionnaires were completed and returned (Table 2.3.2). As expected, distribution through elementary schools was more effective than the mail-out to families of junior and senior high school students. Intercommunity differences in respondent rates were observed ($p < 0.001$). The High exposure community ranked second in the proportion of questionnaires returned. A small sample of nonrespondents (27 to 36) in each community was interviewed by telephone. This sample was adequate to ascertain a demographic profile but too small to yield useful morbidity information. Nonrespondents in the High and Low exposure communities faithfully mirrored the demographic characteristics of respondents. More variation, but no large deviations, were found between respondents and nonrespondents in the two Intermediate exposure areas.

### Table 2.3.2. COMMUNITY CHARACTERISTICS: QUESTIONNAIRE RESPONSE AND RESPONDENT EXCLUSIONS

| Community | Percent of families responding | | Percent of nonasthmatic children excluded | |
|---|---|---|---|---|
| | Distributed through elementary school | Mailed to families of junior and senior high school students | Because of missing data on education, sex, or illness | Because of missing data on age |
| Low | 91 | 34 | 6 | 9 |
| Intermediate I | 83 | 32 | 7 | 9 |
| Intermediate II | 86 | 40 | 7 | 10 |
| High | 88 | 37 | 6 | 8 |
| Total | 87 | 35 | 7 | 9 |

One-sixth of the nonasthmatic children in responding families were excluded because of missing data on sex, educational attainment of the head of household, illness reporting, or age. No children less than 1 year of age were included in the principal analysis, and these accounted for most of the age exclusions. However, such children were included in a special analysis that attempted to relate duration of air pollution exposure to increased lower respiratory illness. Almost identical percentages of respondents were excluded from each community, with the High exposure community having one of the two lowest overall exclusion rates.

Small intercommunity differences were also found in educational attainment ($p < 0.001$). Small, but nonsignificant, community differences were found in mean family size and proportion of parents who were current cigarette smokers (Table 2.3.3). In each case, the High exposure community ranked intermediate when the four study communities were compared. In summary, there was no evidence of intercommunity differences that would bias the results of the survey.

## Asthmatics

As planned in the protocol, asthmatics were excluded from the final analysis of illness. In each area, about 5 percent of the children were reported as having a history of asthma. The number of asthmatics proved too small to permit adequate adjustments for other determinants of acute lower respiratory illness. Except for the High exposure area children, who were reported having significantly higher illness rates for croup, no significant area differences in lower respiratory illnesses or hospitalizations were observed (Table 2.3.4). However, asthmatics exposed to higher levels (9 to 15 $\mu g/m^3$) of suspended sulfates, whether living in the High or Intermediate II exposure areas, tended to report total respiratory illness, pneumonia, bronchitis, and hospitalizations more frequently than asthmatics living in communities with lower sulfate

### Table 2.3.3. COMMUNITY CHARACTERISTICS: EDUCATIONAL ATTAINMENT OF FATHERS, MEAN FAMILY SIZE, AND CURRENT CIGARETTE SMOKING HABITS OF PARENTS

| Community | Percent of fathers who completed high school | Mean family size | Percent of parents who currently smoke cigarettes | |
|---|---|---|---|---|
| | | | Mothers | Fathers |
| Low | 78 | 6.0 | 20 | 31 |
| Intermediate I | 66 | 5.7 | 25 | 37 |
| Intermediate II | 68 | 6.4 | 25 | 38 |
| High | 73 | 6.0 | 22 | 35 |
| Total | 71 | 6.0 | 23 | 35 |

### Table 2.3.4. ASTHMATIC CHILDREN REPORTING AT LEAST ONE LOWER RESPIRATORY ILLNESS DISTRIBUTED BY COMMUNITY AND CLINICAL DIAGNOSIS

| Community | Number of subjects | Percent reporting each respiratory syndrome | | | | |
|---|---|---|---|---|---|---|
| | | Any LRI | Croup | Bronchitis | Pneumonia | Hospitalization |
| Low | 96 | 50 | 29 | 43 | 10 | 10 |
| Intermediate I | 110 | 56 | 24 | 54 | 12 | 12 |
| Intermediate II | 156 | 62 | 30 | 56 | 18 | 17 |
| High | 113 | 60 | 43 | 58 | 15 | 14 |

and sulfur dioxide exposures. Thus, the exclusion of asthmatic children appears to diminish any overall excesses in lower respiratory illness in the High and Intermediate II exposure areas that might later be attributed to sulfur dioxide or suspended sulfate pollution.

## Total Acute Lower Respiratory Illness

Nonasthmatic respondents who comprised the study population of 8991 were then distributed by length of residence within their respective communities (Table 2.3.5). Sample sizes for 2 years of residence were quite small, and were combined with durations of $\leq 1$ year prior to analysis. Younger children experienced more lower respiratory illnesses, with attack rates highest for 2 year olds and steadily decreasing thereafter to a plateau beginning at age 10 (Figure 2.3.1). Average attack rates over the study period showed similar patterns for both sexes. Separate analyses indicated that socioeconomic status was related to illness reporting in a complex fashion. Thus, any analysis relating intercommunity differences in illness to ambient air pollution should isolate or adjust for the effects of age, duration of residence, and socioeconomic status. Data in the following tables reflect such adjustment; unadjusted data are presented in the Appendix, Tables 2.3.A.1 to 2.3.A.5.

The effect of each of the postulated major personal determinants of acute lower respiratory disease (and of each specific lower respiratory disease component considered) was tested. In only 4 of 30 tests was there evidence of deviation from the model assumption, and 3 of these occurred with tests involving croup. Results of the analyses for total acute respiratory illness are given in Table 2.3.6. As expected, age was a powerful determinant of illness frequency, but no consistent sex or socioeconomic effects were found.

Children living in the High exposure area were found to report single and repeated episodes of acute

### Table 2.3.5. NONASTHMATIC CHILDREN USED IN ALL ILLNESS ANALYSES DISTRIBUTED BY COMMUNITY AND RESIDENCE DURATION

| Community | Number of subjects | | | |
|---|---|---|---|---|
| | $\leq 1$ yr residence | 2 yr residence | $\geq 3$ yr residence | Total |
| Low | 153 | 81 | 1719 | 1953 |
| Intermediate I | 236 | 99 | 1968 | 2304 |
| Intermediate II | 253 | 90 | 2161 | 2504 |
| High | 222 | 94 | 1915 | 2231 |
| Total | 864 | 384 | 7763 | 8991 |

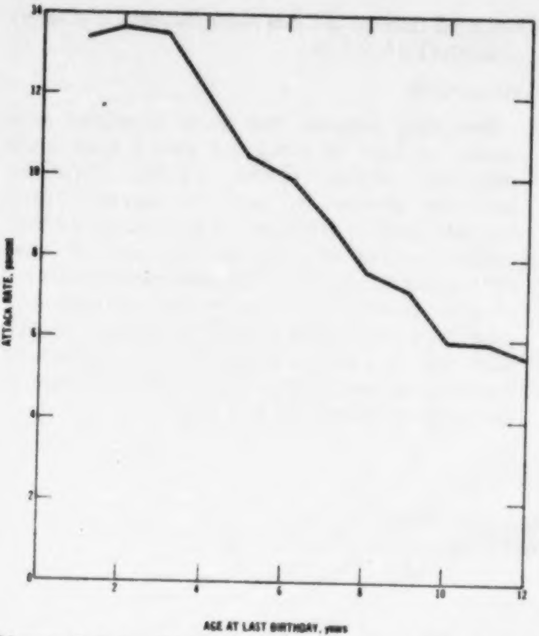

Figure 2.3.1. Age-specific annual mean attack rates for acute lower respiratory illness.

lower respiratory illness significantly more frequently than children living in the other three areas (Table 2.3.7). Such excesses were entirely due to significantly marked increases in reported illness among children residing in the High pollution area for 3 or more years. Although not significant, children living in the High exposure community less than 3 years reported less illness, suggesting that families with exquisitely susceptible children may avoid the High exposure community and that exposures of up to 3 years may be necessary before excess morbidity occurs. There are at least two possible explanations of the low rates in children who have lived in the High exposure community less than 3 years: First, since the presence of chronic respiratory disease symptoms in adults accurately predicted the occurrence of excess lower respiratory disease in their children,[10] perhaps parents of the more susceptible children were prevented from being employed and then moving into the more polluted community by occupational health preplacement screening. Secondly, the High pollution community is small and hence may have fewer endogenous strains of infectious organisms that typically affect migrants than the larger, more heterogenous Low and Intermediate I pollution communities.

Lesser increases also occurred in the Intermediate II exposure community, suggesting that either infrequent peak exposures to sulfur dioxide or long-term exposures to suspended particulate sulfates might also be accompanied by excess acute lower respiratory morbidity.

Table 2.3.6. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, ANY LOWER RESPIRATORY DISEASE

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | <3 yr | ≥3 yr | Any length |
| Pollution exposure | ≥1 | NS | <0.001 | <0.001 |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | <0.05 | NS | <0.10 |
| Pollution exposure | ≥2 | NS | <0.001 | <0.001 |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | NS | NS | NS |

[a] NS—not significant, p >0.10.

## Croup

Laryngotracheobronchitis, or croup, was analyzed in a similar fashion (Table 2.3.8). Croup mimicked the findings already reported for overall lower respiratory illness. Significant increments in single or repeated attacks of croup were especially striking among children residing in the High exposure area for as long as 3 years. As before, new arrivals in the High exposure area actually had less illness than their counterparts in the less polluted communities. No trend towards a morbidity increment was noted in the Intermediate II community, which was exposed to relatively high suspended sulfate levels. Croup was reported significantly more often by the better educated families and was more frequent in younger children (Table 2.3.9).

## Bronchitis

Reporting patterns for acute bronchitis were similar to those of croup and overall acute lower respiratory illness (Table 2.3.10). Significant morbidity excesses in single or repeated illness episodes could be attributed to residence in the High exposure community. Again, these excesses were entirely due to significant increases occurring after 2 years of residence. As before, younger children were more likely to experience single or repeated episodes diagnosed as acute bronchitis, and advantaged families were more likely to report repeated episodes among their children (Table 2.3.11).

**Table 2.3.7. AGE-SEX-SES ADJUSTED 3-YEAR ATTACK RATES FOR ANY LOWER RESPIRATORY ILLNESS BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Community | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | $<3$ yr residence | $\geq 3$ yr residence | Any length residence |
| Low | $\geq 1$ | 25.6 | 27.3 | 27.1 |
| Intermediate I | | 27.8 | 26.5 | 27.0 |
| Intermediate II | | 27.5 | 29.0 | 28.7 |
| High | | 22.9 | 38.2 | 36.1 |
| Low | $\geq 2$ | 19.2 | 15.2 | 15.7 |
| Intermediate I | | 14.6 | 14.6 | 14.6 |
| Intermediate II | | 14.2 | 17.2 | 16.9 |
| High | | 12.3 | 23.4 | 21.9 |

**Table 2.3.8. AGE-SEX-SES ADJUSTED 3-YEAR ATTACK RATES FOR CROUP BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Community | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | $<3$ yr residence | $\geq 3$ yr residence | Any length residence |
| Low | $\geq 1$ | 15.7 | 16.9 | 16.9 |
| Intermediate I | | 11.7 | 14.5 | 14.3 |
| Intermediate II | | 11.7 | 16.5 | 16.3 |
| High | | 9.4 | 26.4 | 24.3 |
| Low | $\geq 2$ | 10.3 | 8.0 | 8.4 |
| Intermediate I | | 5.0 | 5.7 | 5.7 |
| Intermediate II | | 5.0 | 7.8 | 7.5 |
| High | | 4.7 | 13.8 | 12.5 |

### Table 2.3.9. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, CROUP

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | <3 yr | ≥3 yr | Any length |
| Pollution exposure | ≥1 | NS | <0.001 | <0.001 |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | <0.10 | <0.10 | <0.05 |
| Pollution exposure | ≥2 | NS | <0.001 | <0.001 |
| Age | | <0.05 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | <0.10 | <0.10 | <0.05 |

[a] NS—not significant, $p > 0.10$.

### Table 2.3.10. AGE-SEX SES-ADJUSTED 3-YEAR ATTACK RATES FOR BRONCHITIS BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Community | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | <3 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥ 1 | 16.9 | 16.5 | 16.6 |
| Intermediate I | | 18.8 | 16.7 | 17.3 |
| Intermediate II | | 17.6 | 17.1 | 17.3 |
| High | | 16.4 | 23.6 | 22.8 |
| Low | ≥ 2 | 11.3 | 6.5 | 7.0 |
| Intermediate I | | 6.9 | 6.5 | 6.7 |
| Intermediate II | | 8.6 | 7.8 | 8.2 |
| High | | 4.7 | 10.8 | 10.1 |

### Table 2.3.11. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, BRONCHITIS

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | <3 yr | ≥3 yr | Any length |
| Pollution exposure | ≥1 | NS | <0.001 | <0.001 |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | NS | NS | NS |
| Pollution exposure | ≥2 | NS | <0.001 | <0.01 |
| Age | | <0.01 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | <0.05 | <0.01 | <0.05 |

[a] NS—not significant, $p > 0.10$.

## Pneumonia

Single or repeated episodes of physician-diagnosed pneumonitis were infrequent in all study communities (Table 2.3.12). No trends in intercommunity differences attributable to pollution exposure were seen. As expected, pneumonia reporting decreased with age. However, increases in pneumonia bordering on statistical significance were related to lower socioeconomic status (Table 2.3.13).

## Hospitalizations Resulting from Acute Lower Respiratory Illness

Hospitalizations, which were even less frequent than pneumonia, seemed influenced by the same factors as pneumonia (Tables 2.3.14 and 2.3.15).

Table 2.3.12. AGE-SEX-SES ADJUSTED 3-YEAR ATTACK RATES FOR PNEUMONIA BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Community | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | <3 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 4.9 | 4.4 | 4.4 |
| Intermediate I | | 7.7 | 6.5 | 6.8 |
| Intermediate II | | 7.7 | 5.0 | 5.5 |
| High | | 6.0 | 4.8 | 4.9 |
| Low | ≥2 | 3.0 | 1.2 | 1.0 |
| Intermediate I | | 2.8 | 1.3 | 1.7 |
| Intermediate II | | 2.6 | 1.1 | 1.9 |
| High | | 2.0 | 1.2 | 1.1 |

Table 2.3.13. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, PNEUMONIA

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | <3 yr | ≥3 yr | Any length |
| Pollution exposure | ≥1 | NS | NS | NS |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | NS | NS | <0.10 |
| Pollution exposure | ≥2 | NS | NS | NS |
| Age | | NS | <0.001 | <0.001 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | NS | NS | <0.10 |

[a] NS—not significant, p >0.10.

### Table 2.3.14. AGE-SEX-SES ADJUSTED 3-YEAR HOSPITALIZATION RATES BY RESIDENCE DURATION AND NUMBER OF ADMISSIONS

| Community | Number of admissions | Attack rate, percent | | |
|---|---|---|---|---|
| | | <3 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥ 1 | 3.6 | 2.5 | 2.4 |
| Intermediate I | | 4.4 | 2.8 | 3.0 |
| Intermediate II | | 4.9 | 2.1 | 2.6 |
| High | | 4.2 | 2.1 | 2.7 |
| Low | ≥ 2 | 2.9 | 0.4 | 0.5 |
| Intermediate I | | 1.8 | 0.6 | 0.5 |
| Intermediate II | | 2.7 | 0.5 | 0.7 |
| High | | 1.8 | 0.8 | 0.7 |

### Table 2.3.15. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF ADMISSIONS AND RESIDENCE DURATION, HOSPITALIZATION

| Factor | Number of admissions | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | <3 yr | ≥3 yr | Any length |
| Pollution exposure | ≥1 | NS | NS | NS |
| Age | | <0.01 | <0.001 | <0.001 |
| Sex | | NS | NS | <0.10 |
| Socioeconomic status | | NS | <0.05 | NS |
| Pollution exposure | ≥2 | NS | NS | NS |
| Age | | NS | <0.05 | <0.01 |
| Sex | | NS | NS | NS |
| Socioeconomic status | | NS | NS | NS |

[a] NS—not significant, p >0.10.

Recent hospitalizations were more often reported for younger children from less advantaged families. Thus, the increases in acute respiratory illness morbidity attributed to community air pollution, while serious enough to merit physician attention, were unlikely to have been immediately life-threatening. Otherwise, hospitalizations would have paralleled overall morbidity and increased significantly in the more polluted community. Adjusted reporting rates for hospitalizations and pneumonia both tended to be higher in newly arrived children in every study community.

## Age-specific Attack Rates

Selected age-specific lower respiratory illness attack rates adjusted to remove the potentially confounding effects of sex and socioeconomic status were calculated for three age groups (1 to 4, 5 to 8, and 9 to 12) in each study community (Table 2.3.16). Since children of every residence duration were considered, this analysis provided a conservative estimate of intercommunity differences attributable to ambient air pollution exposures. The effects of sex, socioeconomic status, and ambient air pollution were then tested by the linear model previously described, and a probability summary was compiled (Table 2.3.17). Intercommunity differences attributable to the experience of children living in the High exposure community, which were thought to be a manifestation of combined sulfur dioxide-suspended sulfate exposures, could be separated in the analyses from effects that accompanied the overall exposure gradient for suspended particulate sulfates. The expectation was that effects of the lower sulfate (9 $\mu g/m^3$) exposures experienced by children in the Intermediate II community might not be manifest until later in childhood than would effects of the higher sulfur dioxide (91 to 94 $\mu g/m^3$) and

Table 2.3.16. SEX-SES ADJUSTED 3-YEAR AGE-SPECIFIC ATTACK RATES FOR ANY LOWER RESPIRATORY ILLNESS AND BRONCHITIS DISTRIBUTED BY COMMUNITY

| Illness | Community | Attack rate, percent | | |
|---|---|---|---|---|
| | | 1 to 4 yr olds | 5 to 8 yr olds | 9 to 12 yr olds |
| Any lower respiratory illness | Low | 38.6 | 26.6 | 15.7 |
| | Intermediate I | 36.9 | 29.1 | 14.7 |
| | Intermediate II | 37.3 | 30.0 | 19.5 |
| | High | 49.0 | 32.2 | 25.3 |
| Bronchitis | Low | 24.6 | 15.9 | 9.0 |
| | Intermediate I | 24.0 | 17.9 | 9.2 |
| | Intermediate II | 21.4 | 18.6 | 11.9 |
| | High | 30.9 | 19.3 | 17.2 |

Table 2.3.17. PROBABILITY SUMMARIES FOR THE EFFECTS OF POLLUTION EXPOSURE, SEX, AND SOCIOECONOMIC STATUS ON AGE-SPECIFIC ACUTE LOWER RESPIRATORY ILLNESS RATES

| Illness | Age, years | Source of variation[a] | | | |
|---|---|---|---|---|---|
| | | Pollution exposure | | Sex | Socio-economic status |
| | | Sulfur dioxide | Suspended sulfate | | |
| Any lower respiratory illness | 1 to 4 | <0.001 | <0.01 | NS | NS |
| | 5 to 8 | NS | <0.05 | NS | NS |
| | 9 to 12 | <0.001 | <0.001 | NS | NS |
| Bronchitis | 1 to 4 | <0.05 | NS | NS | NS |
| | 5 to 8 | NS | <0.10 | NS | NS |
| | 9 to 12 | <0.001 | <0.001 | NS | NS |

[a] NS—not significant, $p > 0.10$.

sulfate levels (15 $\mu g/m^3$) found in the High exposure community. This was indeed the case. The High exposure community reported more illness at every age when either total acute respiratory illness or acute bronchitis was considered. Intercommunity differences were large in the youngest and oldest children and attenuated in those who were just entering school or kindergarten. Perceptible illness gradients corresponding to the exposure gradient for suspended sulfates were found in the youngest school children and were unmistakable and statistically significant in children over 9 years of age.

There was evidence, then, that 9- to 13-year exposures to annual average suspended sulfate levels of 9 $\mu g/m^3$ may be accompanied by increased respiratory illness. Even though this illness gradient could best be explained by suspended sulfate exposures, there remains a distinct possibility that short-term peak exposures to sulfur dioxide might be responsible. This is unlikely since the most recent monitoring data revealed that 24-hour peak sulfur dioxide exposures during the last quarter of 1970 and the year 1971 were similar in the two Intermediate exposure areas. Similar analyses for croup, pneumonia, and hospitalization were not performed because of restricted sample sizes. In the age-specific rate analysis, the previously recorded weak, yet significant, effects of socioeconomic status were not apparent.

Clinical diagnosis of croup, the lower acute respiratory illness syndrome most likely to show the earliest initial effect on acute lower respiratory illness, was selected to determine the shortest sulfur dioxide-suspended particulate exposure that will induce a significant increment in acute lower respiratory illness. Age-specific attack rates for croup among children whose families lived in the Low and Intermediate I communities for 3 or more years were compared to attack rates for similar children residing in the High exposure area (Figure 2.3.2). In this analysis, the experience of boys and girls was combined to give attack rates greater stability. Without doubt, excess croup was present in the 1 to 2 year old age group. Finer analyses showed an effect in the first year of life. Was the excess in croup caused by short-term peak pollution exposures or longer term annual exposures? This remains a key question that could not be answered by the present retrospective report but may be answered by prospective studies currently in progress. There can be no doubt, however, that exposures to elevated sulfur dioxide (91 $\mu g/m^3$) and suspended sulfate (15 $\mu g/m^3$) levels lasting only a year can be accompanied by significant increases in the frequency of childhood croup.

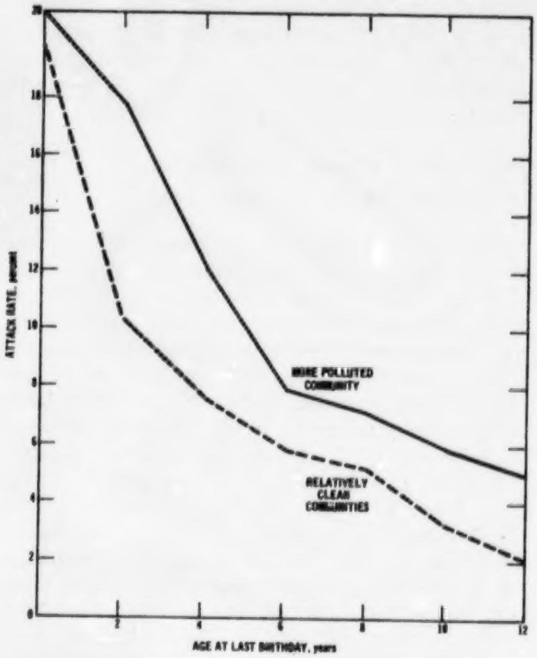

Figure 2.3.2. Age-specific annual mean attack rates for croup distributed by community pollution level.

Age-specific illness rates were also utilized in tests of the significance of indoor air pollution emanating from cigarette smoking by parents. When this was done, the cigarette smoking habits of parents were found to be a significant determinant of acute lower respiratory conditions. The model may have failed to completely separate smoking effects from socioeconomic influences since persons of lower socioeconomic status were more likely to be cigarette smokers. In any case, cigarette smoking within the home was associated with significant increases in acute bronchitis and pneumonia in preschool children age 1 to 4, who are most heavily exposed to home pollution from side-stream cigarette smoke. Unexplained paradoxical effects upon the reporting of croup were found: both families without any smokers and families with heavy smokers reported croup more frequently than families with light smokers. The effects of cigarette smoking, however, could not explain the previously discussed intercommunity differences in childhood acute lower respiratory illness because both the prevalence of smoking and the intensity of cigarette smoking were similar for all communities. Moreover, significant statistical relationships persisted between ambient air pollution and increased reporting of acute bronchitis and croup in analytical models that considered the cigarette smoking habits of parents.

## Diagnostic Patterns and Validation of Questionnaire-reported Illness

Striking intercommunity differences in physician diagnoses for the same set of respiratory symptoms in a young patient could be reflected by significant intercommunity differences in illness reporting by parents who responded to the survey questionnaire. This did not occur. Fifty-three physicians who cared for children comprising the study population were asked to diagnose six respiratory syndromes as either "bronchitis" or "not bronchitis." The symptom complexes in each syndrome were graded ranging from pharyngitis with minimal, but unmistakable, lower respiratory symptoms to classical pneumonia. Bronchitis was purposefully chosen since bronchitis in children is a somewhat imprecise diagnosis. This allowed a better chance to discover any latent intercommunity differences. Physicians and their diagnostic decisions were tallied for each community (Table 2.3.18). Since it was common for physicians to have clinical practices that included children from three of the four study communities, a pool of their answers was tallied. The fact that children from three geographically clustered study communities consulted a common pool of physicians was in itself an unexpected safeguard against diagnostic bias. Physicians from different communities generally had the same diagnostic pattern when considering pharyngitis, classical bronchitis, early pneumonia, or classical pneumonia. Differences were observed when either the mild bronchitis-deep chest cold syndrome or the croup syndrome was considered. Although no significant differences were found, physicians in the Low exposure community were somewhat more likely to call croup bronchitis. Thus, any small degree bias traced to variations in diagnostic customs would be most likely to involve the Low exposure community but not the other three communities.

The questionnaire used in this study classified deep chest colds as bronchitis, which is lower respiratory illness, and this made validation of questionnaire responses a difficult task since physicians will often note only acute respiratory infection or upper respiratory illness when, in fact, they diagnose a chest cold. At times, physicians only record symptoms that are of value in helping recall the patient's medical history. Other possible errors include imperfect memory retention, imprecise doctor-patient communication, and imprecise enumeration. For the validation estimate, either the specific diagnosis of bronchitis or any acute respiratory illness confirmed by medical record was accepted as validation of lower respiratory illness reported by the questionnaire respondents. On the other hand, a questionnaire reporting no illness was not considered in error unless a specific lower respiratory illness syndrome was diagnosed. For each area, a 15 percent sample of questionnaire responses was validated by the described review of medical records. For each physician included in the validation sample, approximately equal numbers of children reported well and sick with bronchitis were selected. (For this reason, the use of sensitivity and specificity measures to arrive at corrected rates is not valid.)[11] Within the sample, attempts were made to validate all lower respiratory illness episodes. The validation effort revealed no

Table 2.3.18. CLINICAL DIAGNOSIS OF ACUTE BRONCHITIS: NUMBER OF PHYSICIANS CHARACTERIZING RESPIRATORY ILLNESS SYNDROMES AS "BRONCHITIS" OR "NOT BRONCHITIS"

| Syndrome description | Low | | Pool of physicians from Int. I, Int. II, and High exposure areas | | Intermediate I | | Intermediate II | | High | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Bronchitis | Not bronchitis | Bronchitis | Not bronchit's | Bronchitis | Not bronchitis | Bronchitis | Not bronchitis | Bronchitis | Not bronchitis |
| Pharyngitis | 0 | 17 | 4 | 32 | 2 | 13 | 1 | 11 | 1 | 8 |
| Deep chest cold or mild bronchitis | 13 | 4 | 18 | 18 | 7 | 8 | 8 | 4 | 3 | 6 |
| Croup or laryngotracheobronchitis | 4 | 13 | 8 | 28 | 3 | 12 | 2 | 10 | 3 | 6 |
| Classical bronchitis | 13 | 4 | 28 | 8 | 11 | 4 | 10 | 2 | | 2 |
| Severe bronchitis or early pneumonia | 8 | 9 | 29 | 17 | 8 | 7 | 7 | 5 | 4 | 5 |
| Pneumonia | 3 | 14 | 5 | 31 | 2 | 13 | 1 | 11 | 2 | 7 |

**Table 2.3.19. VALIDATION OF QUESTIONNAIRE RESPONSES REPORTING CHILDREN EITHER SICK OR WELL AGAINST PHYSICIAN RECORDS[a]**

| | Any lower respiratory illness | | | | Bronchitis | | | |
|---|---|---|---|---|---|---|---|---|
| | Sick validated | | Well validated | | Sick validated | | Well validated | |
| Community | percent | n[b] | percent | n | percent | n | percent | n |
| Low | 70 | 91 | 92 | 85 | 69 | 77 | 96 | 100 |
| Intermediate I | 69 | 108 | 94 | 87 | 69 | 85 | 96 | 114 |
| Intermediate II | 78 | 132 | 79 | 90 | 82 | 109 | 82 | 114 |
| High | 78 | 103 | 84 | 61 | 73 | 86 | 86 | 79 |
| Total | 72 | 434 | 87 | 323 | 74 | 357 | 90 | 407 |

[a] Allowances were made to adjust for multiplicity of diagnostic terms used to describe mixed upper and lower respiratory infections. Otherwise, the agreements would be substantially reduced.

[b] n is the number of records examined.

significant intercommunity differences (Table 2.3.19). Since the High exposure community exhibited high to intermediate values for any of the validating procedures, there was no evidence of bias that could explain intercommunity differences attributed to pollution. Given the diagnostic imprecision and other inherent errors involved, the questionnaire proved to be an adequate, though not perfect, reporting instrument.

## DISCUSSION

Children living in communities characterized by elevated ambient levels of sulfur dioxide and/or suspended particulate sulfate air pollution were reported to have significantly higher attack rates for single or repeated episodes of illnesses grouped as acute lower respiratory disease. Consistent excesses were observed for any acute lower respiratory illness and for croup and bronchitis, two of the most common clinical syndromes within the grouping. No intercommunity differences in episodes of pneumonia or hospitalizations were reported. Morbidity excesses were observed after only 1 year of exposure to elevated levels of both sulfur dioxide and suspended particulate sulfates. More subtle morbidity excesses attributed to suspended sulfate exposures were first apparent after exposures lasting 5 to 8 years and unmistakable after 9 to 12 years.

Asthmatic history, age, socioeconomic status, cigarette smoking in the home, and recent family migration were intervening variables that had a significant effect on the frequency of acute lower respiratory illness. In the present report, the two most powerful determinants of illness, history of asthma and age, were either isolated or adjusted. Weaker covariates, including duration of residence and socioeconomic status, were duly considered in the analysis, as was the effect of parental cigarette smoking. Two other factors that might seriously bias the study were intercommunity differences in the customary diagnostic patterns of physicians serving the study areas and overreporting by respondents in the more polluted communities. Special surveys assured that neither of these had occurred.

Other community characteristics were also closely scrutinized, but none would seem likely to invalidate the study. Smoking patterns, family size, and educational attainment of the head of household were similar for all study communities. Exclusions affected each of the communities equally, and the illness experience of asthmatics who were excluded from the main analysis showed the same morbidity patterns as the nonasthmatics who comprised the principal study population. Samples of nonrespondents from each community were interviewed by telephone and found to exhibit demographic patterns almost identical to those of respondents. Still, there is a remote possibility that nonrespondent illness patterns could differ enough from those of respondents to alter the findings of the study. However, nonrespondents in different communities would have to behave anomalously. Doubling the reporting rate of nonrespondents in Low and Intermediate exposure communities could equalize overall acute lower respiratory illness reporting if nonrespondents in the

High exposure area reported like initial respondents. In the High exposure area, nonrespondents might have the same impact only by reducing their illness reporting to a level one-fifth that of respondents. Such wide variations are unlikely in view of the consistent paucity of other major intercommunity differences involving illness covariates.

One might also choose to question the exposure estimates projected from emissions profiles and dispersion models after calibration with aerometric measurements.[8] Exposure projections, while not precise enough to quantitate short-term peak levels, are most useful tools for the assessment of annual average exposures.

After carefully considering each of the complexities involved, we concluded that significant increases in acute lower respiratory morbidity can be attributed to exposures of 1 or more years characterized by elevated annual average levels of sulfur dioxide (91 $\mu g/m^3$) accompanied by elevated levels of suspended sulfates (15 $\mu g/m^3$) in the presence of low levels of total suspended particulates (62 $\mu g/m^3$), and suspended nitrates (2.0 $\mu g/m^3$). We also found that morbidity excesses accompanied longer (5 to 9 year) exposures to elevated annual average levels of suspended sulfates (9 $\mu g/m^3$) in the absence of elevations of other ambient air pollutants.

The magnitude of the observed morbidity excesses attributed to combined sulfur dioxide and suspended sulfate exposure is worthy of comment (Table 2.3.20). Among nonasthmatic children living as long as 3 years in the High exposure area, overall increases were 42 percent in the proportion of children reporting a single illness and 57 percent in the proportion of children reporting repeated episodes. Morbidity excesses in repeated respiratory illness that were attributable to suspended sulfate pollution of the Intermediate II community were a more modest 15 percent in the absence of high levels of sulfur dioxide. Among asthmatics, the increases in total acute lower respiratory illness (25 percent) and in croup (13 percent) were substantial, but very small increases occurred with every clinical syndrome. In the High exposure community, puzzling, unexplained, statistically insignificant deficits were observed in pneumonia attack rates and hospitalizations.

Reductions in ambient sulfur dioxide concentrations to levels specified by Primary Ambient Air

**Table 2.3.20. EXCESS ACUTE LOWER RESPIRATORY ILLNESS AMONG CHILDREN EXPOSED 3 OR MORE YEARS TO ELEVATED LEVELS OF SULFUR DIOXIDE**

| Illness | Number of episodes reported | Percent excess[a] | | |
|---|---|---|---|---|
| | | In asthmatic children living in High exposure community | Nonasthmatic children living in | |
| | | | Intermediate II community | High exposure community |
| Any lower respiratory illness | ≥ 1 | 25 | 8 | 42 |
| | ≥ 2 | —[b] | 15 | 57 |
| Croup | ≥ 1 | 13 | 6 | 69 |
| | ≥ 2 | —[b] | 15 | 103 |
| Bronchitis | ≥ 1 | 5 | 3 | 42 |
| | ≥ 2 | —[b] | 20 | 66 |
| Pneumonia | ≥ 1 | 3 | —[c] | —[c] |
| | ≥ 2 | —[b] | —[c] | —[c] |
| Hospitalizations | ≥ 1 | 2 | —[c] | —[c] |
| | ≥ 2 | —[b] | 1 | 60 |

[a] Combined experience of Low and Intermediate I communities used as a reference point. Excess risk plus 1.00 equals relative risk.

[b] Not available.

[c] Deficits noted for these categories are based upon relatively rare events.

Quality Standards accompanied by a reduction in suspended sulfates could well significantly reduce acute lower respiratory illness in large numbers of children currently or recently exposed to high pollution levels. However, little is now known about the persistence of induced susceptibility to acute respiratory infection. Repeated surveys, controlled clinical studies, and well designed toxicological studies will be necessary to document and understand the expected improvements in human health.

## SUMMARY

In a survey involving 9000 preschool and elementary school children, the frequency of single or repeated episodes of acute lower respiratory illness significantly increased (40 to 50 percent) after exposures to elevated levels of sulfur dioxide (91 $\mu g/m^3$) and suspended particulate sulfates (15 $\mu g/m^3$) lasting over 2 years. Longer exposures, roughly 9 years, to lower annual average levels of suspended sulfates (9 $\mu g/m^3$) resulted in smaller increments (22 percent) in acute lower respiratory morbidity. As expected, a personal history of asthma and age were the most powerful determinants of acute lower respiratory illness. When parents polluted the home environment by smoking cigarettes, their preschool children more frequently became ill with bronchitis or pneumonia. Socioeconomic factors behaved in a complex fashion. Families with higher social status reported increased croup, increased bronchitis, and decreased pneumonia. Physician diagnostic customs were examined and found similar in all communities. A 15 percent sample of questionnaire responses was validated from physician records, and no intercommunity differences in confirmation of questionnaire responses were noted.

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**Table 2.3.A.1. UNADJUSTED 3-YEAR ATTACK RATES FOR ANY LOWER RESPIRATORY ILLNESS BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | 2 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥ 1 | 28.8 | 14.8 | 24.3 | 24.2 |
| Intermediate I | | 33.5 | 17.2 | 24.8 | 25.4 |
| Intermediate II | | 24.1 | 26.7 | 26.7 | 26.4 |
| High | | 26.6 | 16.0 | 33.8 | 32.4 |
| Low | ≥ 2 | 20.3 | 12.3 | 13.4 | 13.9 |
| Intermediate I | | 18.2 | 11.1 | 13.2 | 13.6 |
| Intermediate II | | 13.8 | 15.6 | 15.3 | 15.2 |
| High | | 14.4 | 9.6 | 21.1 | 20.0 |

**Table 2.3.A.2. UNADJUSTED 3-YEAR ATTACK RATES FOR CROUP BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | 2 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 19.0 | 8.6 | 15.1 | 15.1 |
| Intermediate I | | 15.7 | 9.1 | 13.6 | 13.6 |
| Intermediate II | | 12.6 | 17.8 | 15.1 | 14.9 |
| High | | 12.6 | 9.6 | 23.3 | 21.6 |
| Low | ≥2 | 11.1 | 8.6 | 7.2 | 7.6 |
| Intermediate I | | 5.1 | 6.1 | 5.2 | 5.2 |
| Intermediate II | | 4.7 | 6.7 | 6.9 | 6.7 |
| High | | 4.5 | 6.4 | 12.7 | 11.7 |

**Table 2.3.A.3. UNADJUSTED 3-YEAR ATTACK RATES FOR BRONCHITIS BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | 2 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 20.9 | 4.9 | 14.5 | 14.6 |
| Intermediate I | | 24.6 | 11.1 | 15.2 | 16.0 |
| Intermediate II | | 17.0 | 16.7 | 16.2 | 16.3 |
| High | | 18.5 | 10.6 | 21.5 | 20.7 |
| Low | ≥2 | 13.1 | 3.7 | 6.2 | 6.6 |
| Intermediate I | | 9.3 | 5.1 | 5.8 | 6.2 |
| Intermediate II | | 9.9 | 5.6 | 7.4 | 7.5 |
| High | | 6.3 | 3.2 | 10.2 | 9.5 |

### Table 2.3.A.4. UNADJUSTED 3-YEAR ATTACK RATES FOR PNEUMONIA BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | 2 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 6.6 | 1.2 | 4.2 | 4.2 |
| Intermediate I | | 8.9 | 5.1 | 5.9 | 6.2 |
| Intermediate II | | 7.9 | 4.4 | 4.5 | 4.9 |
| High | | 7.7 | 1.1 | 3.8 | 4.1 |
| Low | ≥2 | 3.9 | 0.0 | 1.3 | 1.5 |
| Intermediate I | | 3.8 | 1.0 | 1.8 | 2.0 |
| Intermediate II | | 2.8 | 1.1 | 1.7 | 1.8 |
| High | | 0.9 | 0.0 | 1.1 | 1.0 |

### Table 2.3.A.5. UNADJUSTED 3-YEAR HOSPITALIZATION RATES BY RESIDENCE DURATION AND NUMBER OF ADMISSIONS

| Community | Number of admissions | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | 2 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 3.3 | 0.0 | 2.2 | 2.2 |
| Intermediate I | | 4.7 | 4.0 | 2.5 | 2.8 |
| Intermediate II | | 6.3 | 4.4 | 1.7 | 2.2 |
| High | | 3.6 | 4.3 | 2.2 | 2.4 |
| Low | ≥2 | 1.3 | 0.0 | 0.2 | 0.3 |
| Intermediate I | | 0.4 | 1.0 | 0.5 | 0.5 |
| Intermediate II | | 2.4 | 1.1 | 0.2 | 0.5 |
| High | | 0.5 | 1.1 | 0.6 | 0.6 |

# 2.4 AGGRAVATION OF ASTHMA BY AIR POLLUTANTS: 1971 SALT LAKE BASIN STUDIES

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## INTRODUCTION

The purpose of this study was to determine what levels of ambient air pollutants are linked with excess illness in asthmatics who comprise 3 to 5 percent of our total population.[1,2] Previous studies have indicated that complex urban air pollutant mixtures aggravate asthma as determined from diaries of patients, emergency room visits, or physician and outpatient records.[3-8] Air pollutants generated by agricultural enterprises and specific types of industrial activity were also found to aggravate asthma.[9-12] Other studies revealed that the effects of meteorological variables, particularly temperature, must be considered in attempts to quantify the role of air pollutants in the aggravation of asthma.[13-18] Unfortunately, there remains a relative deficiency in the dose-response information on the adverse effects of air pollutants upon human health, and such information must provide the basis for National Primary Air Quality Standards. Some of the complexities involved were illustrated by a study that linked air pollutants from a coal-fueled power plant to asthmatic attacks in a dose-response fashion but was unable to disentangle the effects of single pollutants.[19] There remains, then, a real need to define the dose-response functions relating air pollutants to an excessive number of asthmatic attacks.

Western smelter communities are generally located in a fashion that places residential neighborhoods away from the usual path of stack emissions containing high concentrations of sulfur dioxide. On occasion, however, meteorological shifts expose communities to short-term peak pollutant elevations involving stack emissions or particulates that have been resuspended from waste disposal sites. In addition, lower but significant elevations in annual average pollution levels are observed in smelter communities in the absence of high concentrations of suspended particulates, suspended nitrates, nitrogen dioxide, gaseous hydrocarbons, and carbon monoxide. We expected, therefore, that western smelter communities might provide an opportunity to quantify adverse health effects attributable to community air pollution exposures characterized by elevated levels of sulfur dioxide and suspended sulfates.

The diary study reported here comprises the first 26 weeks of surveillance of asthmatics living in Utah. Surveillance began in March 1971 and continued for a total of 67 weeks. The study sought to test the following two hypotheses: first, that excessive asthma attacks among panels of known asthmatic cases would be related to ambient air pollution exposures in a dose-response fashion after removal of the effects of variations in ambient temperature; and second, that any effects attributable to sulfur dioxide and suspended sulfates could be disentangled from the effects of suspended particulates and suspended nitrates.

## MATERIALS AND METHODS

### Selection of the Study Population

Four Salt Lake Basin communities where previously instigated CHESS studies were in progress were used for the asthma study. Located immediately west of the Rocky Mountain Wasatch Range, they are: sections of Ogden and Salt Lake City, Kearns, and Magna. For the other studies, they were selected to represent a pollutant gradient and are thus referred to as Low exposure, Intermediate I, Intermediate II, and High exposure, respectively. The principal point source of sulfur dioxide emissions, a copper smelter, was located approximately 38 miles southwest of Ogden, 13 miles west of Salt Lake City, 8 miles northwest of Kearns, and 5 miles north of Magna. Thus, there would be assurance of some level of sulfur dioxide exposure for all four panels. Unlike other CHESS studies, which compare intercommunity differences, the asthma study will focus on the experience of each community's panel over time.

Asthmatics living in these communities were identified through a School and Family Health Questionnaire administered in December 1970. Additional asthmatics were found by searching the records of community agencies and through the help of local practicing physicians. Each asthmatic was personally interviewed. The interviewer explained the study, elicited consent to participate, administered an asthma panel questionnaire, and taught the asthmatic the diary method for recording daily symptoms. (Examples of the questionnaires and diary form used are given elsewhere.)[20] Asthmatics, diagnosed by a physician, were asked to become panelists after they were interviewed if they gave a history of wheezing and shortness of breath with each asthma attack and if they had two or more attacks during the previous year. Those who agreed to participate lived within a 2-mile radius of an air monitoring station. Highest priority for selection was given to nonsmokers over 16 years of age. The study began with 46 panelists each in the Low exposure and Intermediate I communities, 55 in the Intermediate II community, and 48 in the High exposure community. Replacements for panelists dropped from study were added during the 26 weeks of diary reporting, which began on March 7, 1971, and ended on September 4, 1971.

### Diary Coverage

Each panelist received a diary weekly by mail. He recorded attacks as they occurred each day and returned the completed diary by mail at the end of each week. When instructions for filling out the diary were given by the summer interviewers, each panelist was asked to indicate on the diary that he had an attack whenever he became aware of shortness of breath combined with wheezing. Nonresponse and diaries requiring clarification were investigated by telephone. Diaries received more than 12 days after the last day covered by the diary and diaries that could not be clarified were not accepted for data processing. A log book of diary entries was maintained to permit constant evaluation of panelists' performance. The log book was reviewed weekly to identify over- and underreporting of asthma attacks, which might indicate misunderstanding of what was to be reported, to look for repeated clarification of diaries usually attributable to a misunderstanding of the diary method, and to evaluate frequency of failure to return diaries. Appropriate contact was made with the panelists to improve performance, and continued nonresponders were dropped from the study.

### Monitoring Air Pollution Exposures

Air monitoring stations were installed in each community at a second-story building level approximately 25 feet above the ground. Measurement devices at each station included high-volume samplers for measuring total suspended particulates, dustfall buckets, and sulfur dioxide and nitrogen dioxide bubblers. Continuous 24-hour monitoring was maintained throughout the study period. Sampler filters and bubblers were replaced daily and sent for laboratory analysis. Dustfall buckets were replaced monthly. Minimum and maximum temperatures were recorded at the International Airport that serves the four communities. In the laboratory, sulfur dioxide and nitrogen dioxide gas bubblers were analyzed by methods that are standard for the Environmental Protection Agency, that is, the West-Gaeke and Jacobs-Hochheiser methods, respectively. After dehydration, contents of the dustfall buckets were weighed. High-volume sampler filters were measured gravimetrically and analyzed for sulfate and nitrate fractions. The latter were determined by an automated analysis reduction-diazo coupling method. For the sulfate fraction, a turbidimetric method was used, and turbidity was measured by spectrophotometer. More detailed descriptions of exposure monitoring are presented elsewhere.[21]

### Statistical Analysis

Daily asthma attack rates were computed by dividing the number of panelists who reported one or more asthma attacks on a given day by the number who returned their diaries for that day. This rate computation method was used because some panelists were dropped from the study during the 26 weeks and others were added. Also, some panelists were occasionally negligent in returning their diaries or did not complete diaries when they were temporarily out of the area. Four Intermediate I, four Intermediate II, and three High exposure panelists were not included in the analysis because they failed to return any of the diaries mailed to them.

Data for a single day of the study, then, consisted of the following items: a daily asthma attack rate, a 24-hour average level for each pollutant measured, and a minimum and maximum temperature. These data items for each community were available for each day of the study with the exception of some missing pollutant data. In all analyses, the asthma attack rate was considered the dependent variable. The independent variables were temperature and 24-hour average pollutant levels.

The statistical analysis involved a sequence of five steps: first, plotting weekly averages of asthma attack rates, pollutant levels, and minimal temperature; second, calculating a simple correlation matrix for daily asthma attack rates, pollutant levels, and minimum temperature; third, performing multiple regression analyses to look for pollutant effects after removal of temperature effects; fourth, constructing temperature-specific relative risk and excess risk models for various pollutant levels; and fifth, computing temperature-specific dose estimates to determine, if possible, threshold levels for pollutant effects.[22-24]

To calculate relative risk functions, the total experience of all four panels was combined into a series of temperature-specific relative risk models for appropriate concentrations of each of the pollutants of interest. Since the four panels differed to some extent from each other in the intrinsic frequency of asthmatic attacks, a separate temperature-specific relative risk for each was computed based upon the experience of the panel during the days falling in the lowest concentration range for the particular pollutant of interest. Then, for each panel, relative risks for each subsequent pollutant level in the temperature-specific series were computed using the rate for each successively higher pollutant level as the numerator and the base rate from the lowest pollutant level as the denominator. Relative risks were then weighted by the number of person-days of risk that each represented and combined across communities to achieve a single pooled rate for each temperature-specific pollutant level. The person-days of risk associated with any pooled rate were tallied so that an idea of rate stability might be conveyed. Because the analyses included days on which one or another pollutant might be missing in one or another community, temperature-specific groups of person-days will not balance.

Percentage of excess risk was computed from a base rate. For each of the pollutants of interest, the base rate consisted of the asthma attack rate for warm ($T_{min} \geq 50\,^{\circ}F$), low-pollutant days. Percentage of excess risk was calculated by dividing the appropriate pooled asthma attack rate by the base rate and then subtracting one.

Threshold functions were computed by the method of Hasselblad et al.[23] In setting air quality standards, the existence of a threshold concentration for a health effect is implicitly assumed. It is important, therefore, to ascertain if the dose-response relationship can be reasonably estimated by a function indicating no response until some nonzero threshold concentration is exceeded. Most statistical procedures assume a strictly monotonic functional relationship between two variables, and therefore a significant association exists at all pollutant levels, no matter how low.

As a simple alternative, we hypothesized, for a threshold function, a segmented line having zero slope below exposure level x and with positive slope at levels above x. The point x is estimated by the least squares method. This function has been designated a "hockey stick" function.[23] This least squares technique had been earlier applied to other more general problems.[24]

The estimated threshold, estimated slope, estimated intercept, and the assumption of a linear response above the threshold permitted calculation of an excess risk at the current short-term air quality standard. Multiplying the difference between the standard and the estimated threshold by the estimated slope of the response function provided an estimated asthma attack rate at these allowable once-per-year levels. Percentage of excess risk was calculated by dividing this estimated attack rate by the estimated attack rate intercept and then subtracting one.

## RESULTS

### Environmental Exposures

Pollutant exposure data for 1971 are given in Table 2.4.1. During the spring and summer months covered by the study, aerometric stations in the High and Intermediate II communities reported short-term sulfur dioxide and suspended sulfate exposures that were much higher than the short-term exposures in the Low and Intermediate I communities. This same pattern was not seen for total suspended particulates and suspended nitrates. During the summer quarter, the effect of a strike on sulfur dioxide emissions was reflected by the relatively low median, 90th percentile, and maximum exposures in the High and Intermediate II exposure communities. The experience of panelists during other seasons when peak pollutant exposures are much higher are not covered in this publication, but will be covered in later reports.

One other observation should be emphasized: no single 24-hour exposure during this spring and summer study period exceeded the maximum daily total suspended particulate level (260 $\mu g/m^3$) allowable once yearly under the National Primary Ambient Air Quality Standard, and only 2 days exceeded the similar once-per-year allowable sulfur dioxide standard (365 $\mu g/m^3$). Therefore, adverse effects in this study attributable to either sulfur dioxide or total suspended particulates were within currently allowable air quality limits for a single 24-hour period. Furthermore, none of the study communities exceeded the current National Primary Annual Average Air Quality Standard for total suspended particulates or sulfur dioxide during the study.

### Characteristics of the Study Population

Although the study design did not call for intercommunity comparisons, every attempt was made to

### Table 2.4.1. DAILY AIR POLLUTANT EXPOSURE PATTERNS IN FOUR UTAH COMMUNITIES DISTRIBUTED BY SEASON

| Pollutant and community | 1971 pollutant concentrations, $\mu g/m^3$ | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Median | | | | 90th percentile | | | | Maximum | | | |
| | Spring | Summer | Fall | Winter | Spring | Summer | Fall | Winter | Spring | Summer | Fall | Winter |
| Sulfur dioxide | | | | | | | | | | | | |
| Low | 6 | 4 | 10 | 5 | 17 | 9 | 22 | 18 | 59 | 13 | 51 | 43 |
| Intermediate I | 13 | 7 | 14 | 10 | 31 | 16 | 49 | 34 | 71 | 59 | 259 | 52 |
| Intermediate II | 10 | 4 | 19 | 10 | 47 | 22 | 76 | 52 | 277 | 95 | 358 | 167 |
| High | 64 | 9 | 71 | 46 | 150 | 43 | 189 | 154 | 531 | 91 | 359 | 324 |
| Total suspended particulate | | | | | | | | | | | | |
| Low | 58 | 67 | 69 | 70 | 97 | 99 | 173 | 177 | 223 | 167 | 278 | 276 |
| Intermediate I | 58 | 66 | 80 | 86 | 97 | 87 | 172 | 181 | 198 | 131 | 332 | 258 |
| Intermediate II | 37 | 44 | 34 | 36 | 77 | 59 | 83 | 78 | 157 | 125 | 220 | 134 |
| High | 47 | 51 | 57 | 66 | 88 | 89 | 134 | 136 | 244 | 183 | 482 | 461 |
| Suspended sulfate | | | | | | | | | | | | |
| Low | 4 | 5 | 7 | 4 | 5 | 7 | 12 | 11 | 9 | 13 | 34 | 22 |
| Intermediate I | 5 | 5 | 9 | 6 | 7 | 8 | 22 | 14 | 15 | 19 | 55 | 25 |
| Intermediate II | 5 | 5 | 9 | 6 | 8 | 10 | 23 | 17 | 15 | 23 | 66 | 32 |
| High | 8 | 7 | 13 | 11 | 15 | 18 | 30 | 31 | 28 | 32 | 64 | 66 |
| Suspended nitrates | | | | | | | | | | | | |
| Low | 2 | 3 | 3 | 2 | 3 | 4 | 10 | 5 | 4 | 5 | 25 | 12 |
| Intermediate I | 2 | 3 | 4 | 1 | 4 | 4 | 12 | 7 | 6 | 6 | 31 | 12 |
| Intermediate II | 1 | 2 | 3 | 1 | 3 | 3 | 9 | 4 | 5 | 4 | 47 | 6 |
| High | 1 | 2 | 2 | 1 | 2 | 3 | 10 | 4 | 4 | 4 | 20 | 11 |

select panels as alike as possible. The four panels were similar in size, age, sex, educational attainment, smoking habits, and frequency of asthmatic attacks during the previous year (Table 2.4.2). The Intermediate I community was somewhat different in that fewer panelists were current cigarette smokers. About half of the panelists in each community reported a history of one or more nonasthmatic allergies, including atopic dermatitis, allergic rhinitis, and urticaria. There were, however, no intercommunity differences in the history of such allergic conditions or in the duration of asthma itself.

A difference was noted in turnover among the panels during the study period. In the Intermediate II community, one-third of the original panel withdrew from study by the 26th week, while attrition was 19.5, 23.9, and 16.6 percent for Low, Intermediate I, and High exposure panels, respectively. None of the Low exposure panelists lost were replaced; however, two-thirds of panelists lost from the other panels were replaced with new panelists. These differences are important because new panelists are usually characterized by a tendency to overreport during their first month of participation. Three clusters of newly recruited panelists were identified: four panelists were added during week 15 to the Intermediate I panel, seven during week 15 to Intermediate II panel, and five during week 16 to the High exposure panel. Examination of diary recordings of panelists added to Intermediate I and II panels showed they had reported very few asthma attacks. The five new High exposure panelists, however, reported a significant number of attacks.

The overall response rate for the four panels was good. Five-sixths of the almost 5000 diaries mailed were useable in the analysis. Weekly response rates varied somewhat for any particular week in all four communities.

### Temporal Patterns

An overall impression of the relationship between the frequency of a disorder and factors that might influence that disorder may be gained by scrutiny of temporal patterns. Though we expected that short-term 24-hour pollutant exposures, not weekly exposures, would be most likely to aggravate asthma, weekly averages of attack rates, minimum daily temperature, and selected pollutants were nevertheless computed and plotted to gain a knowledge of seasonal trends. Experiences of all four communities were evaluated, but only plots for the Low and High exposure communities are presented for illustrative purposes.

When average weekly asthma attack rates and average daily minimum temperature for the same week were plotted together, asthma rates in both the High and Low exposure communities were seen to decrease as temperature increased. Weekly asthma attack rates were, however, more erratic than weekly temperature averages (Figure 2.4.1). Asthma rates were lowest in both communities during late summer, weeks 18 to 22 of the study. Attack rates for the Intermediate II and High exposure communities

Table 2.4.2. CHARACTERISTICS OF ASTHMA DIARY STUDY POPULATION

| Community | Population characteristics | | | | | |
|---|---|---|---|---|---|---|
| | Total number of subjects (N) | Subjects >16 years of age, percent | Female subjects, percent | ≥high school completed by household head, percent | Adults currently smoking, percent | ≥10 asthma attacks during past year, percent |
| Low | (46) | 61 | 52 | 80 | 25 | 37 |
| Intermediate I | (49) | 49 | 51 | 67 | 17 | 45 |
| Intermediate II | (66) | 42 | 46 | 85 | 39 | 41 |
| High | (50) | 56 | 46 | 74 | 36 | 38 |
| Total | (211) | 51 | 48 | 77 | 30 | 40 |

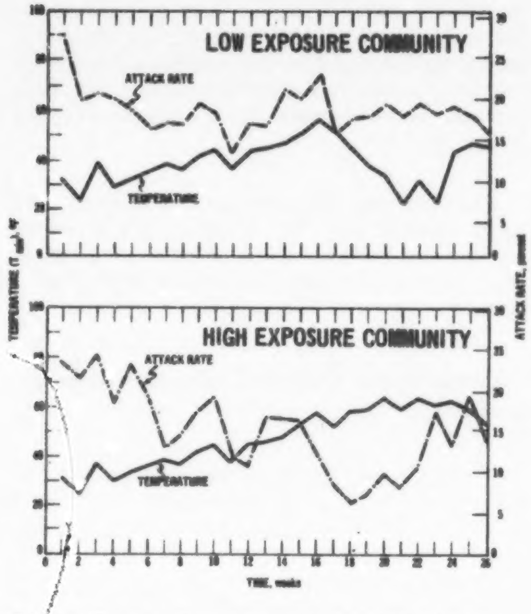

Figure 2.4.1. Weekly mean asthma attack rates compared with weekly average minimum temperatures.

dropped to their summer lows 2 weeks earlier than the other two communities.

Weekly average sulfur dioxide, total suspended particulate, and suspended sulfate air pollutants were each in turn plotted in concert with weekly average asthma attack rates. Sulfur dioxide levels in the Low exposure community were quite low, except for one peculiar week during summer, and generally had no discernible relationship to either asthma attacks or the previously described temperature trend (Figure 2.4.2). The High exposure community was quite different. During March and April (weeks 1 to 9), the coldest weeks of the study, there was an apparent inverse relationship between sulfur dioxide levels and asthma attack rates, similar to the pattern seen in Figure 2.4.1 for temperature and asthma attack rates. Thus, the effect of temperature could be masking the effect of sulfur dioxide during these cold months. Levels of sulfur dioxide in the High exposure community reached zero levels during weeks 18 to 20 due to a smelter strike in July.

Total suspended particulates were then considered in the same fashion (Figure 2.4.3). No clear relationship between particulates and asthma was seen for

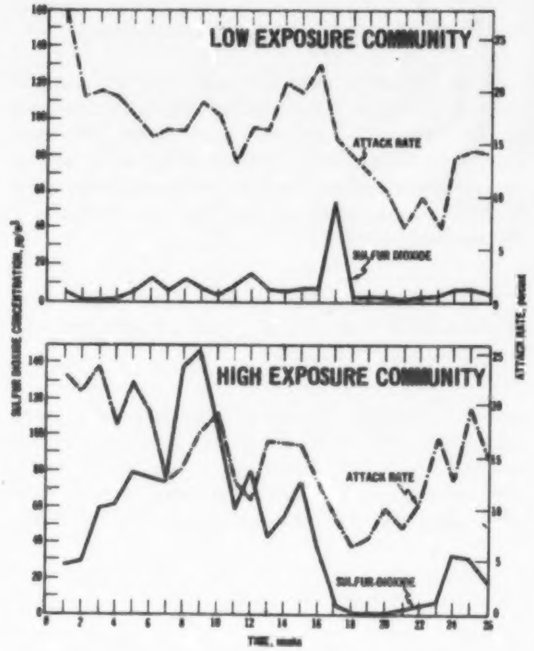

Figure 2.4.2. Weekly mean asthma attack rates compared with weekly mean sulfer dioxide levels.

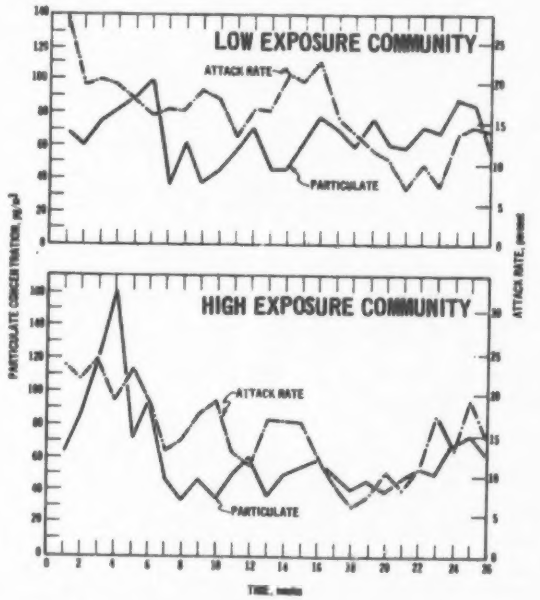

Figure 2.4.3. Weekly mean asthma attack rates compared with weekly mean total suspended particulate levels.

either community, with the possible exception of the last 10 weeks in the High exposure community.

Next, a similar plot for suspended sulfates was evaluated (Figure 2.4.4). In the High exposure community, suspended sulfate levels followed the same general trend noted for asthma attack rates; weekly average suspended sulfate levels were consistently low in the Low exposure community, and unrelated to fluctuations in asthma.

The temporal pattern of suspended nitrates (not illustrated) seemed related to the seasonal temperature trend but not to asthma.

The nitrogen dioxide concentrations were calculated and were included in the statistical analyses. These analyses indicated that nitrogen dioxide levels in the four areas were relatively low. Their temporal pattern was related to that of suspended nitrates and temperature, but as was the case for suspended nitrates, was not strongly related to asthma fluctuations. However, recent reevaluation by the Environmental Protection Agency indicates that the measurement method for nitrogen dioxide is subject to interferences and variable collection efficiencies. Therefore, the nitrogen dioxide data, while still summarized elsewhere,[21] are not included in this report.

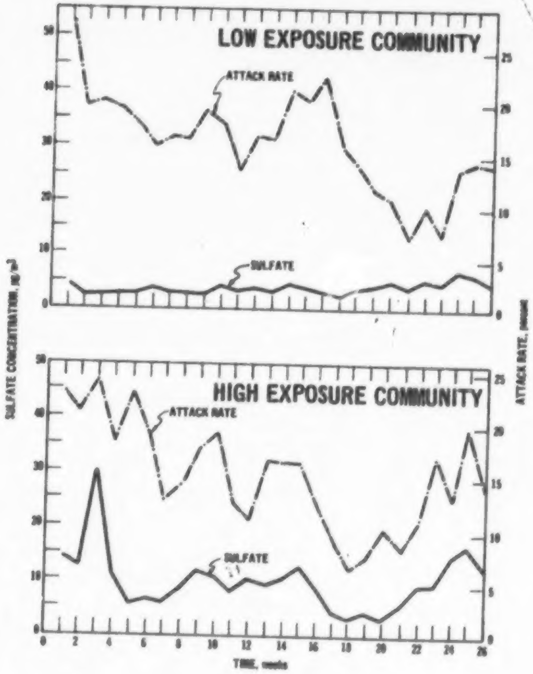

Figure 2.4.4. Weekly mean asthma attack rates compared with weekly mean sulfate fraction levels.

Based on the temporal patterns analyzed, we could conclude only that temperature and perhaps one or more pollutant might be influencing asthma attack rates and that suspended sulfates might play an important role in the High exposure community.

## Correlation and Stepwise Multiple Regression Analysis

To gain a better view of the complex tangle of interrelated factors, a simple correlation matrix was constructed (Table 2.4.3). Temperature was unquestionably the strongest and most consistent apparent determinant of asthma attack rates, with asthma frequency decreasing as temperature increased.

Suspended nitrates seemed at first glance to exert the greatest effects upon asthma. One might falsely conclude that decreasing nitrate air pollution actually increased asthma. Since nitrates were present in low levels and were strongly and positively correlated with temperature, it seemed probable that the negative correlations only reflected a temperature effect.

The negative correlation of sulfur dioxide with temperature was statistically significant in the High exposure community. The suspended sulfate correlation with temperature, although also negative, was not statistically significant. It is probable that any deleterious effects exerted by these pollutants were masked by the temperature effect. Furthermore, sulfur dioxide and suspended sulfates seemed too closely correlated to allow later separation.

There were no consistent correlations between total suspended particulates and any of the other pollutants. In relation to temperature, total suspended particulates were either negatively or not at all correlated.

Hopefully, one could separate the effects of total suspended particulates and suspended nitrates from each other and from those of sulfur dioxide or suspended sulfates. Stepwise multiple regression techniques were employed to separate definite pollutant

**Table 2.4.4. SUMMARY OF MULTIPLE REGRESSION ANALYSES CONSIDERING THE EFFECT OF MINIMUM TEMPERATURE ON ASTHMA ATTACK RATE, FOLLOWED BY THE EFFECTS OF AIR POLLUTANT LEVELS**

| | Source of variation[a] | | | | |
|---|---|---|---|---|---|
| | | Temperature plus: | | | |
| Community | Temperature alone | Sulfur dioxide | Total suspended particulates | Suspended sulfates | Suspended nitrates |
| Low | <0.001 | <0.10 | NS | NS | <0.10 |
| Intermediate I | <0.001 | NS | NS | NS | NS |
| Intermediate II | <0.001 | NS | <0.05 | NS | NS |
| High | <0.001 | NS | <0.02 | <0.005 | NS |

[a] NS—not significant, $p > 0.10$.

should focus primarily on two pollutants (particulates and suspended sulfates) and also on sulfur dioxide to define the relative and excess risks attributable to each and to establish, if possible, an appropriate mathematical dose-response model, whether linear, threshold, or curvilinear.

## Temperature-specific Risk Models

By computations described in the "Materials and Methods" section, relative risks were calculated for the pollutants of interest (Table 2.4.5). The strongest effect by far was noted for suspended sulfates at warmer temperatures ($T_{min} \geq 50$ °F); 24-hour levels as low as 6.1 $\mu g/m^3$ were linked to an increased relative risk for asthma. Similar findings were obtained when the temperature divide was set at $T_{min} = 40$ °F. The inverse relationship observed for temperature and asthma attack rates in the High exposure community could be masking the true extent of the sulfate effect. One can be confident that the sulfate effect was not influenced spuriously by sulfur dioxide; the sulfate effect could be seen in the Low and Intermediate I communities where the influence of modest levels of sulfur dioxide could not be discerned in the asthma attack rates.

The effect of total suspended particulates seemed independent of temperature. Roughly equal increments in attack rates occurred at the same total suspended particulate levels in either temperature range.

The effect of low daily minimum temperature ($T_{min} = 30$ to 50 °F) obviated or obscured any aggravation of asthma caused by 24-hour sulfur dioxide levels of <365 $\mu g/m^3$. Since sulfur dioxide levels in the High exposure community decreased as temperature increased and since asthma attack rates follow the opposite temperature trend, one cannot be sure that temperature is still not hiding a true sulfur dioxide effect.

We concluded that suspended particulates and suspended sulfates at relatively low levels exerted an independent aggravating effect on the frequency of asthma attacks. Any effect of low levels of sulfur dioxide that might have occurred at higher ambient temperatures could not be separated with certainty from the effect of suspended sulfates.

Excess risk due to the effect of temperature or air pollution fluctuations on the frequency of asthma attack rates were determined from appropriate excess risk functions as described in the "Materials and Methods" section (Table 2.4.6). The base rate for suspended sulfates computed when $T_{min} \geq 50$ °F at a level of $\leq 6$ $\mu g/m^3$ of the pollutant was 10.8. There was an increasing excess risk of asthma attacks with increasing levels of suspended sulfate when temperatures were $\geq 50$ °F. At the higher levels of suspended sulfates ($\geq 10.1$ $\mu g/m^3$) the excess risk computed was 50 percent. An approximately equal excess risk (53 percent) was found in the 30 to 50 °F temperature range at low levels of the pollutant ($\leq 6$ $\mu g/m^3$).

This pattern was not seen for either total suspended particulates or for sulfur dioxide. Instead

### Table 2.4.5. POLLUTANT AGGRAVATION OF ASTHMA AS QUANTIFIED BY TEMPERATURE-SPECIFIC RELATIVE RISK MODELS

| Pollutant | Minimum temperature, °F | Integrated 24-hour concentration, μg/m$^3$ | Relative risk[a] | Person-days of observation[b] |
|---|---|---|---|---|
| Sulfur dioxide | 30 to 50 | $\leq 60$ | 1.00 (16.9) | 13,871 |
| | | 61 to 80 | 0.70 | 942 |
| | | 81 to 365 | 0.93 | 1,576 |
| | | $>365$ | 0.81 | 100 |
| | $\geq 50$ | $\leq 60$ | 1.00 (12.4) | 12,847 |
| | | 61 to 80 | 1.16 | 672 |
| | | 81 to 365 | 1.12 | 216 |
| Total suspended particulates | 30 to 50 | $\leq 60$ | 1.00 (16.1) | 11,789 |
| | | 61 to 75 | 1.12 | 1,940 |
| | | 76 to 244 | 1.15 | 3,663 |
| | $\geq 50$ | $\leq 60$ | 1.00 (12.1) | 9,734 |
| | | 61 to 75 | 1.08 | 3,930 |
| | | 76 to 244 | 1.17 | 3,207 |
| Suspended sulfates | 30 to 50 | $\leq 6$ | 1.00 (16.6) | 13,212 |
| | | 6.1 to 8 | 0.95 | 1,946 |
| | | 8.1 to 10 | 0.85 | 894 |
| | | 10.1 to 28 | 0.89 | 1,346 |
| | $\geq 50$ | $\leq 6$ | 1.00 (10.8) | 10,816 |
| | | 6.1 to 8 | 1.17 | 3,094 |
| | | 8.1 to 10 | 1.35 | 965 |
| | | 10.1 to 32 | 1.50 | 1,957 |

[a] Base rate for temperature-specific relative risk is in parentheses.

[b] Person-days for pollutants may not balance across temperature-days because one or more pollutants may be missing on one or more days.

both showed highest excess risk on cooler days (30 to 50 °F) at the lower pollutant levels and approximately one-half to one-third less excess risk than this on warm days ($T_{min} \geq 50$°F) at pollutant levels of $\geq 61$ μg/m$^3$.

## Threshold and Linear Dose-response Functions

Threshold functions were fitted for sulfur dioxide, total suspended particulates, and suspended sulfates to determine if there is a safe exposure below which pollutant-induced aggravation in asthma will not occur or if a linear dose-response function can be established so that one can balance socially acceptable control measures with a socially acceptable increase in asthma attacks.

Threshold functions for sulfur dioxide, considering two warm temperature ranges ($T_{min} \geq 40$ °F and $T_{min} \geq 50$ °F), are plotted in Figure 2.4.5. In either case, the best estimate was that an effect began at a concentration near zero, but there was a reasonable probability (0.05) the threshold might be as high as 23 to 54 μg/m$^3$ (Figure 2.4.5). The steeper slope belongs to the set of warmer days.

At the present 24-hour once-a-year allowable sulfur dioxide level of 365 μg/m$^3$, the threshold functions predict that asthma attack rates would

**Table 2.4.6. RELATIVE EFFECT OF TEMPERATURE AND AIR POLLUTANTS ON ASTHMA ATTACK RATES AS QUANTIFIED BY EXCESS RISK FUNCTIONS**

| Pollutant | Minimum temperature | Pollutant concentration, $\mu g/m^3$ | Excess risk of asthma,[a] percent |
|---|---|---|---|
| Sulfur dioxide | ≥50 °F | ≤60 | 00 (12.4) |
| | 30 to 50 °F | ≤60 | 36 |
| | ≥50 °F | 61 to 80 | 16 |
| | ≥50 °F | 81 to 365 | 12 |
| Total suspended particulates | ≥50 °F | ≤60 | 00 (12.1) |
| | 30 to 50 °F | ≤60 | 33 |
| | Either range | 61 to 75 | 10 |
| | Either range | 76 to 260 | 16 |
| Suspended sulfates | ≥50 °F | ≤ 6 | 00 (10.8) |
| | 30 to 50 °F | ≤ 6 | 53 |
| | ≥50 °F | 6.1 to 8.0 | 17 |
| | ≥50 °F | 8.1 to 10 | 35 |
| | ≥50 °F | ≥10.1 | 50 |

[a] Base rate for excess risk is in parentheses.

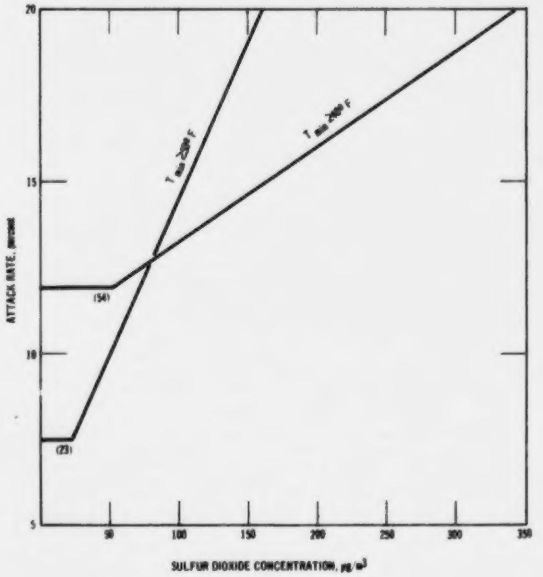

Figure 2.4.5. Effect of minimum daily temperature and sulfur dioxide on daily asthma attack rates.

increase by 51 percent based on cooler days ($T_{min} \geq 40$ °F) and by 254 percent based on warmer days ($T_{min} \geq 50$ °F) (Table 2.4.7). If the allowable once-per-year level were lowered, for example, to 335 $\mu g/m^3$, the concomitant reduction in expected excess illness would be modest, at best a one-tenth reduction in the asthma excess. On colder days ($T_{min} < 40$ °F) no sulfur dioxide effect can be ascertained.

Threshold functions could also be established for excess asthma attributable to total suspended particulates for two temperature ranges ($T_{min}$ = 30 to 50 °F and $T_{min} \geq 50$ °F) in the Low and Intermediate I communities (Figure 2.4.6 and Table 2.4.7). As expected from the temperature-specific relative risk analyses, the theoretical threshold for the lower temperature range (107 $\mu g/m^3$) is higher than the threshold for the higher range (71 $\mu g/m^3$). In both cases, 24-hour suspended particulate levels now allowable once per year (260 $\mu g/m^3$) might be expected to more than double asthma attack rates.

Figure 2.4.6 also shows that the threshold levels for total suspended particulates containing an elevated suspended sulfate component were identical (26 $\mu g/m^3$) for the two temperature ranges con-

Table 2.4.7. TEMPERATURE-SPECIFIC THRESHOLD ESTIMATES FOR THE EFFECT OF SELECTED POLLUTANTS ON ASTHMA ATTACK RATES

| Pollutant | Minimum daily temperature, °F | Intercept (attack rate, percent) | Estimated effects threshold, $\mu g/m^3$ | Slope | Estimated percent excess risk at presently allowed once-per-year level |
|---|---|---|---|---|---|
| Sulfur dioxide | ≥40 | 12.0 | 54 | 0.000198 | 51 |
| | ≥50 | 80.0 | 23 | 0.000595 | 254 |
| Total suspended particulates without high sulfates | 30 to 50 | 14.8 | 107 | 0.001141 | 118 |
| | ≥50 | 9.8 | 71 | 0.000882 | 170 |
| Total suspended particulates with high sulfates | 30 to 50 | 17.1 | 26 | 0.000440 | 60 |
| | ≥50 | 11.0 | 26 | 0.000686 | 146 |
| Suspended sulfates | 30 to 50 | 16.0 | 17.4 | 0.005500 | 9[a] |
| | ≥50 | 9.8 | 1.4 | 0.003274 | 62[a] |

[a] Since no standard exists for suspended sulfates, the level of 20 $\mu g/m^3$, which is now found on 5 to 10 percent of days in urban areas, was arbitrarily chosen to illustrate the excess risk.

sidered. For the currently allowable once-a-year 24-hour particulate standard of 260 $\mu g/m^3$, an excess in asthma attacks of 60 percent for the lower temperature range and 146 percent for the higher range might be expected. Clearly, the chemical composition of suspended particulates is a critically important determinant of their biological effects. Substantial new efforts are required to define and control particulate pollution.

A last threshold function was calculated for suspended sulfates (Figure 2.4.7 and Table 2.4.7), and the threshold level for the higher temperature range ($T_{min} \geq 50$ °F) was very low (1.4 $\mu g/m^3$) while the level for cooler days ($T_{min}$ = 30 to 50 °F) was higher (17.4 $\mu g/m^3$). At levels of 20 $\mu g/m^3$, which are found on 5 to 10 percent of the days in our large cities, asthma rates could be expected to increase by one-tenth for the lower temperature range and by three-fifths for the higher range.

## DISCUSSION

The data reported here suggest association of excess asthma attacks with several complex environmental influences. Because of their preliminary nature, they must be interpreted with caution. These first 26 weeks of study covered the period March 7 through September 4, 1971, the greater part of which is the summer season and the known period of lowest pollution in the Salt Lake Basin. Thus, during this period, effects of pollution would be less amenable to identification and measurement. In addition, problems of interferring factors, such as airborne allergens, were probably present during the summer months of this initial part of the study.

The possible effect of seasonal peaks in plant allergens presents a problem that is broad in scope and difficult to define. Various methods have been used to identify and quantitate pollens, none of which have been altogether successful. Efforts are presently in progress to develop a method for measuring protein content of ambient air. Until feasible and reliable measurement technology is available, the problems of assessing the effects of airborne allergens are prohibitive. In the presently reported study, the probable net effect of such agents would have been to make associations between pollutants and asthma more difficult to establish. Any allergen peak that happened to coincide with a high pollutant exposure peak in any of the four communities would probably also influence asthma

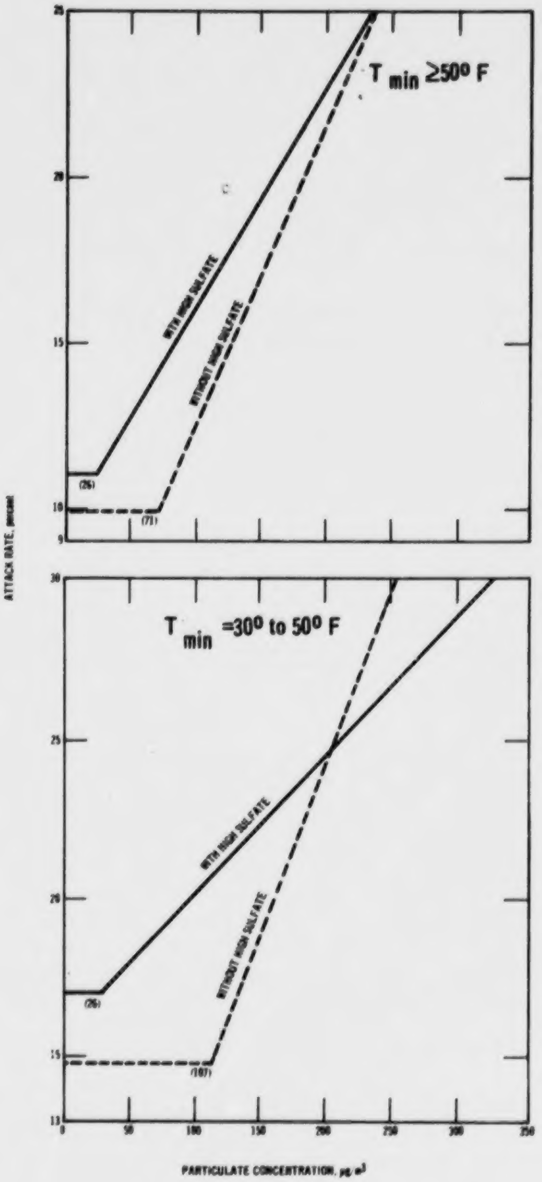

Figure 2.4.6. Effect of total suspended particulates with and without a high sulfate content on asthma attack rate.

attack rates in the other nearby study communities, increasing asthma attack rates for all panels and possibly obscuring true differences attributable to ambient air pollution.

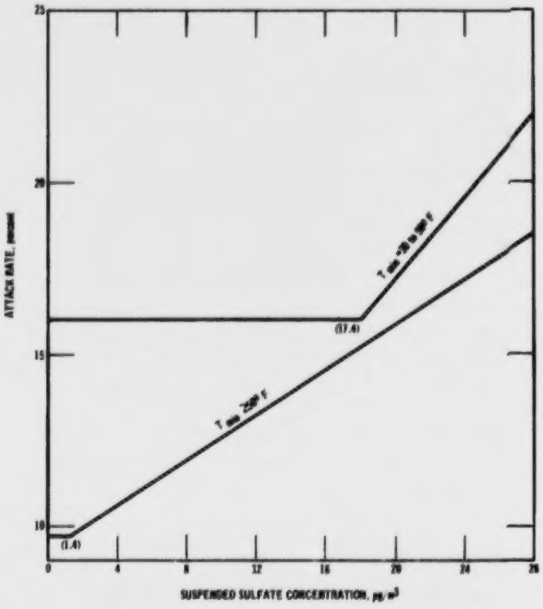

Figure 2.4.7. Effect of minimum daily temperature and suspended sulfates on daily asthma attack rates.

We also considered possible sources of bias inherent in the population studied. Exclusion of some panelists from the study was of first importance. Every effort was made to locate all possible asthmatics living within the monitoring range. Only those diagnosed as asthmatics by a physician at some time in their lives were accepted. This exclusion of medically undiagnosed persons was felt justified because of the nature of asthma; the frightening pulmonary syndrome makes it unlikely that persons subject to acute asthma attacks would fail to seek medical care. Another small group excluded were those who would not agree to participate because they planned to move or claimed no recent asthma attacks.

Initially, nonsmokers and adults were to receive selection priority, but the number of asthmatics in the communities was so limited that none were excluded for smoking habits or age. A second source of bias that must be considered, then, is the influence of smoking cigarettes. An unexpected result of a finer analysis was an inconsistently high asthma attack rate found for nonsmokers compared with the rates of smokers. However, this observation is tenuous as the smoker rate was based on very small numbers. We speculate, however, that the severity of asthma in the nonsmokers was greater, causing them to stop smoking or to be lifetime nonsmokers. The habit of smoking could not bias the study since each panel had a roughly equal proportion of smokers.

Finally, the addition of panelists in clusters during the study must be considered as a possible source of bias. Clustering of new panelists occurred in three communities. In Intermediate I and II this clustering had no effect on attack rates because of the few attacks reported by the new panelists. Attacks reported by a cluster of five panelists added in the High exposure community during week 16 had the effect of reducing pollutant impact on attack rates because they were added at a point in time when the attack rate of this community was at its lowest level.

An unusual event that occurred during July was a strike involving the major source of sulfur dioxide emissions. The shut-down of operations by the strike was accompanied by a pronounced improvement in air quality and a reduction in asthma attack rates that occurred sooner and were larger than seasonal reductions observed in the more distant study communities some 2 weeks later. Thus, a reduction in pollution similar to that which might result from stringent pollution control action measurably benefited asthmatics, although it undoubtedly had a major adverse economic impact.

Analysis of environmental measurements linked specific air pollutants with excess asthma attacks. Clearly, low ambient temperature was found to be the strongest determinant. This finding is consistent with previous studies conducted by other investigators.[1,19,25] The effect of low temperature was apparent during the first 8 weeks of surveillance when weekly means ranged between 24 and 39 °F. One might expect a further increase in mean attack rates attributable to the low ambient temperatures of Utah's winter during the remainder of the 67-week study.

Temporal patterns and simple correlation analysis were clouded by the powerful temperature effect but provided clues pointing to specific pollutants, most importantly the effects of suspended sulfates. The intervening strong temperature variable could be isolated and adjusted for in further analyses. Multiple regression results showed a temperature effect in every community and revealed a significantly high association of suspended sulfates after the temperature effect was removed. Significant residual effects were also seen in the two most polluted communities for total suspended particulates.

The temperature effect was further delineated by the relative and excess risk models. Suspended sulfates and sulfur dioxide effects for low levels of the pollutants were reflected in increased relative risks for asthma on warm days and an increase in relative risk on both warm and cold days for exposure to higher levels of total suspended particulates. Excess risks could be measured for all three of these pollutants.

After considering all facets of the study, we concluded that increased asthma attacks could be related to 24-hour exposures involving modestly elevated levels of suspended particulate matter (71 $\mu g/m^3$) during warm weather ($T_{min} \geq 50$ °F) and higher elevations (107 $\mu g/m^3$) in colder weather ($T_{min}$ = 30 to 50 °F). Secondly, we felt it prudent to consider the threshold level for sulfur dioxide on warm days ($T_{min} \geq 40$ °F) to be 23 to 54 $\mu g/m^3$. Finally, we concluded that the threshold for aggravation of asthma by suspended sulfates was 1.4 $\mu g/m^3$ on warm days ($T_{min} \geq 50$ °F) and 17.4 $\mu g/m^3$ on cooler days ($T_{min}$ = 30 to 50 °F).

These results suggest that achieving the present primary air quality standards will not necessarily protect asthmatics from excess illness attributable to ambient air pollution. Excess asthma attributable to sulfur dioxide might be expected on 5 to 10 percent of summer days. Excesses attributable to total suspended particulates could occur on up to 5 percent of summer days and 30 percent of fall and winter days. Most importantly, excesses due to suspended sulfates are likely to occur on 10 percent of fall and winter days and 90 percent of summer days. Suspended sulfates tend to penetrate through suburban residential rings into more rural areas, thus exposing a much larger population. Demonstrated pollutant-induced excesses in asthma are an important public health problem since asthma ranks as one of the leading causes of chronic illness. Relatively small increases in asthma attack rates could easily involve several million extra days of disability.

Analysis of the complete 67 weeks of this study, now in progress, will permit comparison of seasonal variation in factors of biological origin, varying meteorologic factors, and differing levels and combinations of the air pollutants. Data from the 1973 study conducted by the same methods and in these same communities will offer further opportunity to evaluate the effects of pollutants over a greatly extended period of time.

## SUMMARY

Four panels, totaling 211 asthmatics, reported asthma attacks using weekly diaries for 6 months during 1971. Excess asthma attack rates could be attributed to colder days and to ambient air pollutants. Total suspended particulates and suspended sulfates had significant effects at minimum temperature ranges greater than 30 °F. The strongest effects were attributed to suspended sulfates; the most consistent effects were caused by suspended particulates; and the least impressive effects could be attributed to sulfur dioxide exposures for the higher temperature range considered ($T_{min} \geq 50$ °F). The threshold estimates for the lower temperature range ($T_{min}$ = 30 to 50°F) were 107 $\mu g/m^3$ for total suspended particulates and 17.4 $\mu g/m^3$ for suspended sulfates. For the higher temperature range, the threshold levels were 23 to 54 $\mu g/m^3$ for sulfur dioxide, 71 $\mu g/m^3$ for total suspended particulates, and 1.4 $\mu g/m^3$ for suspended sulfates. The importance of chemical composition in determining effects thresholds was seen in the comparison of the threshold level for suspended particulates containing high sulfate with that for particulates with relatively low sulfate content (26 and 71 $\mu g/m^3$, respectively).

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# CHAPTER 3
# ROCKY MOUNTAIN STUDIES

# 3.1 HUMAN EXPOSURE TO AIR POLLUTANTS IN FIVE ROCKY MOUNTAIN COMMUNITIES, 1940-1970

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## INTRODUCTION

Western smelting communities usually have little pollution from automobiles or from nonsmelting industrial sources. Ambient air pollution in these communities is characterized by high levels of both sulfur dioxide and total suspended particulates. These levels are considered high since they are in excess of National Primary Air Quality Standards. Since smelters represent an approximate point source of pollutants, estimates of previous community exposures to both sulfur dioxide and particulates can be made through the use of production data.

Health studies of respiratory morbidity in these communities should help to distinguish the effects of simple mixtures and of air pollutants like sulfur dioxide, total suspended particulates, and suspended sulfates from the effects of more complex urban mixtures. Such scientific studies can be used to support National Primary Air Quality Standards that will adequately protect the public health and yet will not unnecessarily penalize the economy. In November 1970, the frequencies of lower respiratory disease in children through age 12 and of chronic respiratory disease symptoms in their parents were surveyed retrospectively in five Rocky Mountain communities.[1,2] This report provides environmental exposure information for these health studies and reports the recent history of ambient exposure to sulfur dioxide, total suspended particulates, and suspended sulfates in the five communities. The present survey was a sequel to the Helena Valley Area Environmental Report.[3]

## COMMUNITY DESCRIPTION

Five communities were selected on the basis of air quality data published by the States of Idaho and Montana and advice from health department officials in both states.[4-8] Kellogg is in the Idaho panhandle, and Anaconda, East Helena, Helena, and Bozeman are in Montana (Figure 3.1.1). Kellogg is about 250 miles northwest of Helena and East Helena; Helena, Anaconda, and Bozeman form an isosceles triangle, with all three sides being approximately 100 miles in length.

### Bozeman, Montana

Bozeman, with a population of about 18,500, is essentially an agricultural community, the site of Montana State University, and the county seat of Gallatin County. Bozeman is about 4800 feet above sea level and is located in a large valley bounded on the east by the Bridger Mountains and on the south by the Madison Range. The Gallatin River flows about 7 miles east of the community. There are no large manufacturing industries in the area. A number of inversions have been noted in the Bozeman area, but the air pollution is quite low.[7]

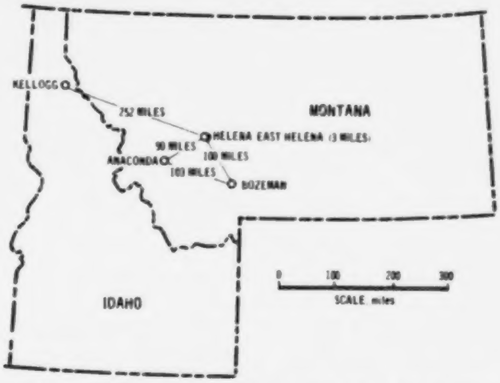

Figure 3.1.1. Location of the five Rocky Mountain study communities.

### Helena-East Helena, Montana

Helena and East Helena are about 3 miles apart in the Helena Valley and have populations of about 23,000 and 1700, respectively, according to the 1970 U. S. Census. The valley is about 25 miles north to south and 35 miles east to west. Helena, with an average elevation of 4100 feet, is located on the south side of this intermountain valley, which is bounded on the north and east by the Big Belt Mountains and the west and south by the main chain of the Continental Divide. The southern parts of the city have an elevation of about 3800 feet. The average height of the mountains above the valley floor is about 3000 feet. There are no large manufacturing concerns located in the city. Helena is the state capital and the county seat of Lewis and Clark County. Since Helena residents live upwind of the smelter in East Helena, they are only intermittently exposed to ambient air pollutants emitted by the smelter (Figure 3.1.2).

East Helena, which has been the site of a lead smelter since 1888, also has a zinc fuming plant, a zinc and talc processing plant, and offices and retail business establishments. Virtually all East Helena

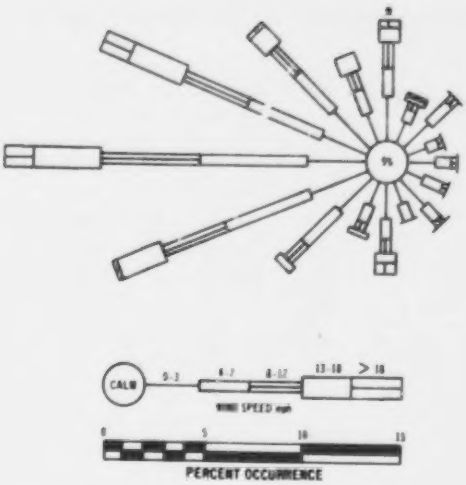

Figure 3.1.2. Surface climatologic wind rose, Helena Municipal Airport, November 1949-October 1954.

residents do their major shopping in Helena, and some of them work in Helena. The elevation of East Helena is about 3900 feet, and the ground slope is much less than that of Helena. The ground south of East Helena, where the smelting operations are located, is 30 to 50 feet higher than the city.[6] East Helena residents also live upwind of the smelter and are thus only occasionally exposed to the emitted air pollutants.

## Anaconda, Montana

Anaconda, with a population of about 9800, is situated just north and west of the Continental Divide at an elevation of about 5300 feet. The mountains rise abruptly at the south side of the city. Anaconda is at the southern end of the Deer Lodge Valley, which is approximately 35 miles long and 8 to 10 miles wide, sloping in a northerly direction from Anaconda to Garrison (4500 feet). Anaconda is the county seat of Deer Lodge County and has been the site of a large copper smelter since the turn of the century.[4,5] The smelter processes copper and zinc ores from Anaconda mines and does no custom smelting. The present 585-foot stack was built in 1902 and is almost 1500 feet above the valley floor because it rests upon a tall slag heap. Electrostatic precipitators were installed in 1917 and have been upgraded technologically about every 2 years since then. Since 1955, the company has used ores from open pit mines, which are lower in sulfur content than the underground ores that were used previously. Since 1965, some sulfur has been metallurgically eliminated. Hoods were also installed on the converters in 1966 to reduce sulfur dioxide emissions. An acid plant, which will be completed in the near future, is expected to reduce sulfur dioxide emissions to about 300 tons/day.

## Kellogg, Idaho

Kellogg has a population of about 3800 and is in the famous Couer d'Alene mining district of the Idaho panhandle. Kellogg sits at the base of a narrow canyon on the west slopes of the Bitterroot Mountain Range at an elevation of about 2300 feet. The Bunker Hill Company complex in Kellogg consists of a mine, an ore concentrator, a smelter, a zinc plant, a fertilizer plant, and a research and analytical laboratory. The Bunker Hill smelter has been a custom lead and zinc smelter since the turn of the century. Approximately two-thirds of its ores are from a wide variety of domestic and foreign mines of other companies. The zinc plant stack is 250 feet high and is about 810 feet above the valley floor. The smelter stack is 200 feet high and is about 337 feet above the valley floor.

## Climatology

The climate for all five communities may be described basically as modified continental. Winter temperatures are affected by Artic cold waves and occasionally may drop well below zero. Summertime temperatures are moderate, with maximum readings generally under 90 °F and very seldom reaching 100 °F. Total precipitation varies from approximately 10 inches in the semiarid northern portion to 30 inches in the humid areas along the Continental Divide. Most of the precipitation occurs from May to July, the rest of the year being relatively dry. Snow may fall from September through May. Cold air drains into the valleys from surrounding mountain slopes at night. Hence, strong and persistent temperature inversions are common throughout this area of Idaho and Montana.

## ESTIMATING HUMAN EXPOSURE TO AIR POLLUTANTS

In the two nonsmelter communities, Bozeman and Helena, long-term exposure to sulfur dioxide, total suspended particulates, and suspended sulfates was based on recently measured values.[3,6,9] Prior exposure was assumed to have been similar to recent exposure since there were no known large changes in either community emissions or meteorologic patterns. Estimates of long-term exposure to sulfur dioxide, total suspended particulates, and suspended sulfates in the three smelter communities, East Helena, Kellogg, and Bozeman, were based upon annual metal production data, estimates of stack emissions of both particulates and sulfur dioxide, and observed air quality measurements.

## Metal Production and Emission Controls

Annual metal production estimates for each smelter[10-13] were supplemented with actual production data and sulfur dioxide and particulate emissions data provided to the U. S. Environmental Protection Agency by each corporation.[14-16] A summary of the annual copper, lead, and zinc production data for East Helena, Anaconda, and Kellogg for the years 1940-1970 is given in Table 3.1.1. Production figures

**Table 3.1.1. ANNUAL PRODUCTION OF COPPER, LEAD, AND ZINC IN THREE ROCKY MOUNTAIN SMELTER COMMUNITIES, 1940-1970[10-13, 16]**

| Year | Production, tons/year | | | |
|---|---|---|---|---|
| | East Helena lead | Anaconda copper | Kellogg | |
| | | | Lead | Zinc |
| 1970 | 57,721 | 120,412 | 123,106 | 95,637 |
| 1969 | 45,343 | 103,314 | 123,986 | 105,700 |
| 1968 | 42,824 | 69,480 | 124,134 | 102,946 |
| 1967 | 50,160 | 65,483 | 122,247 | 92,134 |
| 1966 | 67,133 | 128,061 | 113,194 | 90,000 |
| 1965 | 65,019 | 115,489 | 93,753 | 90,990 |
| 1964 | 69,926 | 103,806 | 86,282 | 91,760 |
| 1963 | 63,693 | 79,762 | 93,430 | 81,296 |
| 1962 | 62,433 | 94,021 | 93,961 | 76,756 |
| 1961 | 58,650 | 104,000 | 107,857 | 74,735 |
| 1960 | 74,412 | 91,972 | 31,658 | 26,449 |
| 1959 | 37,882 | 65,911 | 91,940 | 61,191 |
| 1958 | 69,310 | 90,683 | 91,769 | 55,454 |
| 1957 | 67,517 | 91,512 | 103,420 | 68,832 |
| 1956 | 62,133 | 96,426 | 99,816 | 57,799 |
| 1955 | 58,774 | 81,542 | 88,126 | 56,625 |
| 1954 | 72,694 | 59,349 | 82,520 | 47,404 |
| 1953 | 72,144 | 77,617 | 69,635 | 54,037 |
| 1952 | 60,265 | 61,948 | 57,220 | 54,340 |
| 1951 | 61,033 | 57,406 | 52,501 | 54,468 |
| 1950 | 79,394 | 54,478 | 73,294 | 53,922 |
| 1949 | 64,817 | 56,611 | 43,414 | 41,853 |
| 1948 | 55,217 | 58,252 | 58,150 | 42,067 |
| 1947 | 57,737 | 57,900 | 51,038 | 41,801 |
| 1946 | 28,097 | 58,481 | 33,707 | 34,833 |
| 1945 | 39,995 | 88,506 | 37,974 | 33,110 |
| 1944 | 49,463 | 117,856 | 52,638 | 36,563 |
| 1943 | 59,232 | 138,295 | 53,265 | 41,129 |
| 1942 | 67,010 | 140,465 | 67,389 | 39,916 |
| 1941 | 70,197 | 128,712 | 65,739 | 39,285 |
| 1940 | – | 129,071 | 54,642 | 37,471 |

for the American Smelting and Refining Company (Asarco) at East Helena and for the Bunker Hill Corporation at Kellogg were taken from the yearbooks of the American Bureau of Metal Statistics.[10-13] In the case of the Anaconda Corporation, the yearbooks give total values for smelting performed at Anaconda, Montana; Miami, Arizona; and Hayden, Arizona. In order to estimate the amount of smelting at the Anaconda plant at Anaconda, Montana, figures for the total copper produced in the state of Montana from domestic ores were used. These production figures may be slightly low because they do not include the ore that Anaconda obtains from Canada. For example, in 1971, 7339 tons of ore, matte, and regulous copper were imported into the United States from Canada. If the assumption were made that all of these imports were smelted in Anaconda, Montana, this would only increase the estimated production by approximately 7 percent.

Sulfur dioxide and particulate emission factors for copper smelting, lead smelting, and zinc smelting[17] were applied to the annual metal production figures in order to determine average daily stack emissions of both sulfur dioxide and particulates. Estimates of stack emissions for both particulate and sulfur dioxide for East Helena for the years 1941-1970 were provided by Asarco. Estimated sulfur emissions for the Bunker Hill plant in Kellogg were provided for the years 1940, 1945, 1950, 1955, 1959, 1965, 1970, and 1971 by the Bunker Hill Corporation. Estimates of particulate emissions for the years 1970 and 1971 were also provided by the same company. Estimates of particulate emissions for the years 1965 and 1971 were provided by the Anaconda Corporation. Sulfur dioxide emission measurements were provided by Anaconda for the year 1971. These emissions were compared with those determined by application of emission factors. On the basis of these comparisons, estimates of pollution control efficiencies were made for the three areas.

At the Anaconda smelter, electrostatic precipitators were installed in 1917 and upgraded technologically about every 2 years. Anaconda's precipitators are presently estimated to be 90 percent efficient. It is quite reasonable to assume this particulate emission control efficiency for the period 1940-1970. In the case of sulfur dioxide, the data provided indicate a 23 percent control of sulfur dioxide emissions for the years 1965-1971. Since 1955, pit mined ores, which have less sulfur concentration than vein mined ores, have been used. It is difficult to estimate the actual controls during the period 1940-1964 because of the change in both the type of ore used and the metallurgical process. An approximate average control of 11 percent for sulfur dioxide emissions was assumed for this period.

Since sulfur emissions data are available for 8 years in Kellogg, better estimates of sulfur dioxide emission controls can be made. The evidence indicates approximately 50 percent control during the period 1959-1971, 43.5 percent control during the period 1955-1958, and no control during the period 1940-1954. In the case of particulate emissions, the estimated factors are 97.5 percent control from 1968-1971, 95 percent control from 1965-1967, and 90 percent control from 1940-1964. Summaries of estimated sulfur dioxide and particulate emissions for East Helena, Anaconda, and Kellogg during the period 1940-1970 are shown in Tables 3.1.2 and 3.1.3.

### Exposure Estimates for East Helena

By comparing observed air quality data to average daily pollutant emissions from a given isolated point source, one can estimate the ratio of annual average pollutant concentration to the average rate of pollutant emission from the point source. In the case of East Helena, annual average total suspended particulate concentrations are available for the years 1966, 1967, 1968, and 1969. The ratios of observed annual average total suspended particulate concentration to average daily particulate emitted are shown for these years in Table 3.1.4. The average ratio is 1.05 ($\mu g/m^3$)/(ton/day). The range of the ratios is 0.50. Using the techniques of inefficient statistics described by Dixon and Massey,[18] the standard deviation of the data was estimated to be 0.24. Using the scientific convention of expressing variation at the 50 percent confidence level, the ratio of total suspended particulate concentration to tons of particulate emitted per day was estimated to be 1.05 ± 0.16 ($\mu g/m^3$)/(ton/day).

Similar date for sulfur dioxide are also given in Table 3.1.4. Using the same analysis, the ratio of the concentration of sulfur dioxide to tons of sulfur dioxide emitted per day was estimated to be 0.33 ± 0.10 ($\mu g/m^3$)/(ton/day). These ratios were multiplied by the appropriate daily emissions of sulfur dioxide and particulates (Tables 3.1.2 and 3.1.3) to obtain estimated annual average concentrations of total suspended particulate and sulfur dioxide for the time period 1941-1971. The results of these calculations for East Helena are given in Table 3.1.5. These total suspended particulate and sulfur dioxide estimates are

### Table 3.1.2. ESTIMATED AVERAGE DAILY SULFUR DIOXIDE EMISSIONS IN THREE ROCKY MOUNTAIN SMELTER COMMUNITIES, 1940-1970[10-17]

(tons/day)

| | $SO_2$ emissions | | |
|---|---|---|---|
| Year | East Helena | Anaconda | Kellogg |
| 1970 | 239 | 635 | 262 |
| 1969 | 221 | 545 | 266 |
| 1968 | 153 | 367 | 262 |
| 1967 | 104 | 346 | 245 |
| 1966 | 128 | 675 | 233 |
| 1965 | 118 | 609 | 219 |
| 1964 | 116 | 629 | 212 |
| 1963 | 113 | 483 | 203 |
| 1962 | 108 | 570 | 200 |
| 1961 | 120 | 630 | 140 |
| 1960 | 143 | 558 | 67 |
| 1959 | 71 | 399 | 164 |
| 1958 | 115 | 550 | 220 |
| 1957 | 120 | 555 | 260 |
| 1956 | 112 | 584 | 234 |
| 1955 | 113 | 495 | 219 |
| 1954 | 146 | 360 | 291 |
| 1953 | 148 | 471 | 289 |
| 1952 | 116 | 375 | 267 |
| 1951 | 116 | 348 | 259 |
| 1950 | 139 | 330 | 285 |
| 1949 | 102 | 334 | 205 |
| 1948 | 108 | 353 | 332 |
| 1947 | 108 | 352 | 218 |
| 1946 | 61 | 355 | 166 |
| 1945 | 95 | 537 | 181 |
| 1944 | 108 | 714 | 205 |
| 1943 | 111 | 838 | 220 |
| 1942 | 119 | 852 | 242 |
| 1941 | 125 | 781 | 237 |
| 1940 | – | 783 | 219 |

### Table 3.1.3. ESTIMATED AVERAGE DAILY PARTICULATE EMISSIONS IN THREE ROCKY MOUNTAIN SMELTER COMMUNITIES, 1940-1970[10-17]

(tons/day)

| | Particulate emissions | | |
|---|---|---|---|
| Year | East Helena | Anaconda | Kellogg |
| 1970 | 0.323 | 8.9 | 3.21 |
| 1969 | 0.304 | 7.6 | 3.41 |
| 1968 | 0.298 | 5.1 | 3.36 |
| 1967 | 0.219 | 4.8 | 6.24 |
| 1966 | 0.298 | 9.5 | 5.97 |
| 1965 | 0.304 | 8.5 | 5.66 |
| 1964 | 0.323 | 7.8 | 5.52 |
| 1963 | 0.328 | 5.9 | 5.25 |
| 1962 | 0.271 | 7.0 | 5.05 |
| 1961 | 0.246 | 7.7 | 3.84 |
| 1960 | 0.287 | 6.8 | 1.70 |
| 1959 | 0.095 | 4.9 | 4.33 |
| 1958 | 0.263 | 6.7 | 4.23 |
| 1957 | 0.367 | 6.8 | 4.89 |
| 1956 | 0.304 | 7.1 | 4.32 |
| 1955 | 0.520 | 6.0 | 4.05 |
| 1954 | 0.441 | 4.4 | 7.20 |
| 1953 | 0.583 | 5.7 | 7.22 |
| 1952 | 0.353 | 4.6 | 6.75 |
| 1951 | 0.413 | 4.2 | 6.57 |
| 1950 | 0.564 | 4.0 | 7.35 |
| 1949 | 0.479 | 4.2 | 5.23 |
| 1948 | 0.583 | 4.3 | 5.77 |
| 1947 | 0.994 | 4.3 | 5.52 |
| 1946 | 0.846 | 4.3 | 4.26 |
| 1945 | 1.139 | 6.5 | 4.22 |
| 1944 | 1.084 | 8.7 | 2.17 |
| 1943 | 0.646 | 10.2 | 2.20 |
| 1942 | 0.452 | 10.4 | 2.78 |
| 1941 | 0.301 | 9.5 | 2.72 |
| 1940 | – | 9.5 | 2.77 |

presented graphically in Figures 3.1.3 and 3.1.4 in order to indicate the relative precision of the estimates.

Suspended sulfate data for East Helena are available. During the period 1965-1966, 66 measurements of suspended sulfate indicated an annual average concentration of 7.9 $\mu g/m^3$. The observed total suspended particulate during this period of time was 96 $\mu g/m^3$. Hence the ratio of suspended sulfate observed to particulate observed during this period was 0.082. A series of 25 measurements made during the time period April to August 1968 indicated a suspended sulfate to total suspended particulate ratio of 0.057. In order to estimate the range of this ratio, measurements that were made during the time period June through October 1969 at two stations were compared. These measurements indicated a suspended sulfate to total suspended particulate ratio range of 0.037. Using the statistical technique described previously, the ratio of suspended sulfate to total suspended particulate for East Helena was estimated to be 0.063 ± 0.022 $(\mu g/m^3)/(\mu g/m^3)$. By multiplying this factor by the total suspended

**Table 3.1.4. RATIO OF POLLUTANT CONCENTRATIONS TO POLLUTANT EMISSIONS FOR EAST HELENA, MONTANA**

| Pollutant and year | Emissions, tons/day | Observed annual average concentration, $\mu g/m^3$ | Ratio |
|---|---|---|---|
| Total suspended particulate | | | |
| 1969 | 111 | 136 | 1.23 |
| 1968 | 109 | 97 | 0.89 |
| 1967 | 80 | 104 | 1.30 |
| 1966 | 109 | 87 | 0.80 |
| Sulfur dioxide | | | |
| 1971 | 264 | 78 | 0.296 |
| 1970 | 239 | 52 | 0.217 |
| 1969 | 221 | 104 | 0.470 |

particulate concentrations in Table 3.1.5, estimates were made of the suspended sulfate concentrations for East Helena during the period 1940-1970. The results of these calculations are also given in Table 3.1.5. A graphical summary of the estimates of the suspended sulfates concentrations for East Helena is shown in Figure 3.1.5.

Examination of Table 3.1.5 shows that the estimates of annual average total suspended particulate for East Helena were consistently above the National Primary Air Quality Standard of 75 $\mu g/m^3$ for an annual geometric mean.[19] For example, during the time period 1968-1970, average total suspended particulate concentration was estimated to be 115 $\mu g/m^3$. Similarly, the estimated average total suspended particulate concentration was 146 $\mu g/m^3$ during the decade 1950-1959, and 270 $\mu g/m^3$ during the 9-year period 1941-1949.

In contrast to these high total suspended particulate concentrations, the sulfur dioxide levels in East Helena (Table 3.1.5) have been lower than the National Primary Air Quality Standard (80 $\mu g/m^3$ annual arithmetic mean).[19] For example, during the time period 1968-1970, the average sulfur dioxide concentration was estimated to be 67 $\mu g/m^3$. During the period 1964-1967, an even lower level of sulfur dioxide concentration, 38 $\mu g/m^3$, was estimated. The estimated annual average had also been low during the decades of the forties and fifties. Suspended sulfate estimates (Table 3.1.5) were 7.3 $\mu g/m^3$ during the period 1968-1970, and 9.2 $\mu g/m^3$ during the decade of the fifties. At present there is no National Primary Air Quality Standard for suspended sulfates.

**Table 3.1.5. ESTIMATED ANNUAL AVERAGE CONCENTRATIONS OF SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE FOR EAST HELENA, 1941-1970**
**($\mu g/m^3$)**

| Year | Pollutant | | |
|---|---|---|---|
| | $SO_2$ | TSP | SS |
| 1970 | 78.9 | 123.8 | 7.8 |
| 1969 | 72.9 | 116.5 | 7.3 |
| 1968 | 50.5 | 114.2 | 7.2 |
| 1967 | 34.3 | 83.9 | 5.3 |
| 1966 | 42.2 | 114.2 | 7.2 |
| 1965 | 38.9 | 116.5 | 7.3 |
| 1964 | 38.3 | 123.8 | 7.8 |
| 1963 | 37.3 | 125.7 | 7.9 |
| 1962 | 35.6 | 103.9 | 6.5 |
| 1961 | 39.6 | 94.3 | 5.9 |
| 1960 | 47.2 | 110.0 | 6.9 |
| 1959 | 23.4 | 36.4 | 2.3 |
| 1958 | 38.0 | 100.8 | 6.4 |
| 1957 | 39.6 | 140.7 | 8.9 |
| 1956 | 37.0 | 116.5 | 7.3 |
| 1955 | 37.3 | 199.3 | 12.6 |
| 1954 | 48.2 | 169.1 | 10.7 |
| 1953 | 48.8 | 223.4 | 14.1 |
| 1952 | 38.3 | 135.3 | 8.5 |
| 1951 | 38.3 | 158.3 | 10.0 |
| 1950 | 45.9 | 216.2 | 13.6 |
| 1949 | 33.7 | 183.6 | 11.6 |
| 1948 | 35.6 | 223.4 | 14.1 |
| 1947 | 35.6 | 381.0 | 24.0 |
| 1946 | 20.1 | 324.2 | 20.4 |
| 1945 | 31.4 | 436.5 | 27.5 |
| 1944 | 35.6 | 415.4 | 26.2 |
| 1943 | 36.6 | 247.6 | 15.6 |
| 1942 | 39.3 | 173.2 | 10.9 |
| 1941 | 41.3 | 115.4 | 7.3 |

## Exposure Estimates for Kellogg

Two years of data for total suspended particulate were available for Kellogg, Idaho.[20,21] During 1970, the total suspended particulate concentration averaged 107 $\mu g/m^3$. During 1971, the corresponding value was 92 $\mu g/m^3$. By comparing these values with the particulate emissions for Kellogg in Table 3.1.3, an estimate can be made of the ratio of total suspended particulate concentration to particulate

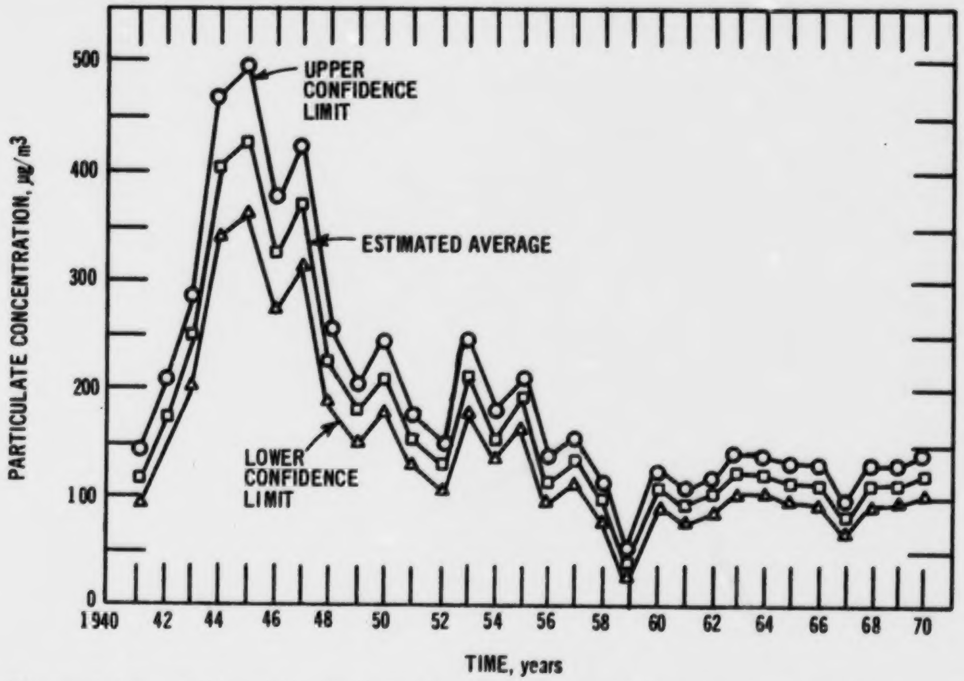

Figure 3.1.3. Estimated annual average total suspended particulate concentrations versus time for East Helena.

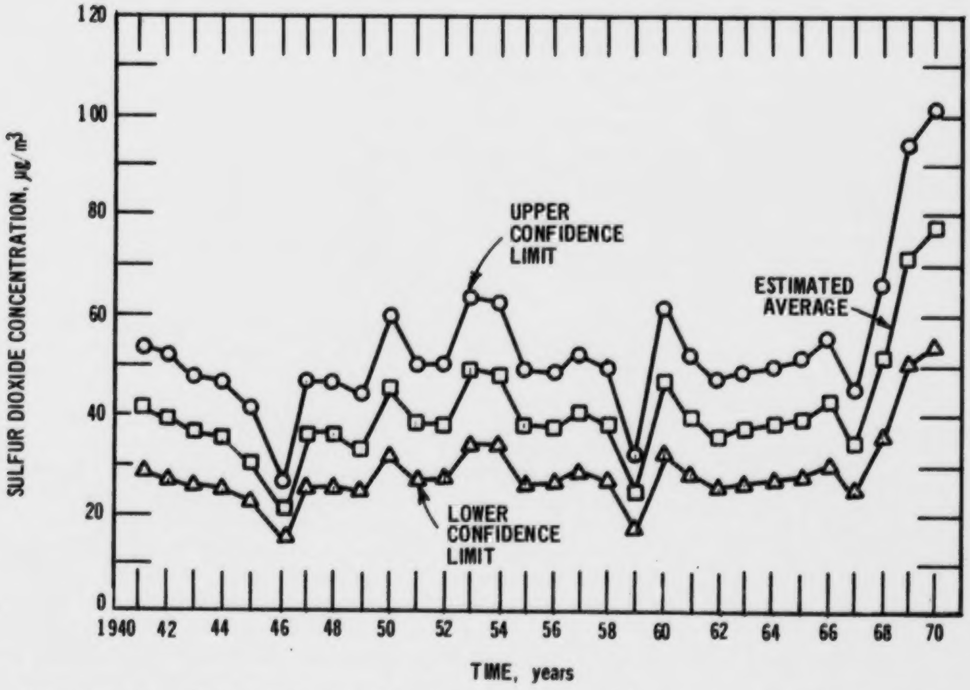

Figure 3.1.4. Estimated annual average sulfur dioxide concentrations versus time for East Helena.

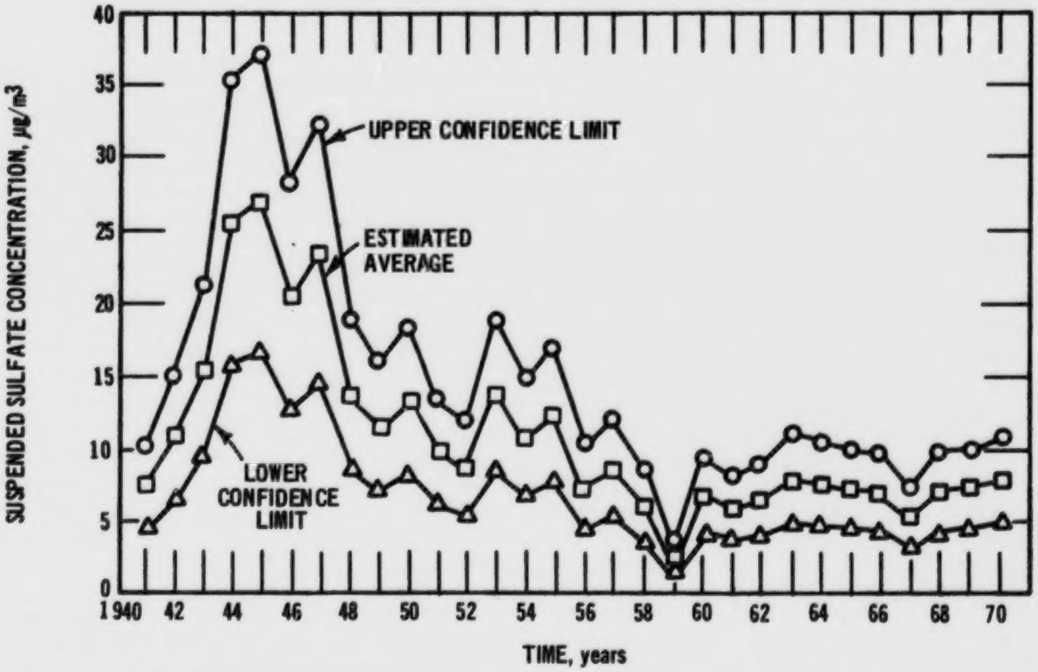

Figure 3.1.5. Estimated annual average suspended sulfate concentrations versus time for East Helena.

emission using the approach outlined for East Helena. For Kellogg, this results in a ratio of total suspended particulates to particulate emittants of 30.6 ± 2.7 (μg/m³)/(ton/day). Historical estimates of total suspended particulate concentration are made by multiplying the particulate emissions in Table 3.1.3 for Kellogg by this ratio. These estimated total suspended particulate concentrations are shown in Table 3.1.6.

Two years of air quality data are available for sulfur dioxide in Kellogg. During 1970, an average sulfur dioxide level of 419 μg/m³ was observed. During 1971, the corresponding value was 359 μg/m³. Comparing these observed values with the appropriate average sulfur dioxide emissions in Kellogg, the ratio of sulfur dioxide concentrations to emissions was estimated to be 1.63 ± 0.21 (μg/m³)/(ton/day). The estimates of sulfur dioxide exposure in Kellogg (Table 3.1.6) during the period 1940-1970 were made by multiplying this factor by the sulfur dioxide emissions shown in Table 3.1.2. The estimates indicated that sulfur dioxide concentrations in Kellogg were very high. For example, during the period 1968-1970, an average value of 374 μg/m³ was estimated. During the decades of the forties and fifties, ambient sulfur dioxide levels were estimated to be 316 and 353 μg/m³, respectively.

No observed values of suspended sulfate were available for Kellogg. Estimates of suspended sulfate were made by comparison with the other smelter communities, Magna and East Helena. Data observed for Magna during the period January 1971-June 1972 indicated an average ratio of suspended sulfate concentration to total suspended particulate of 0.159.[22] The available data for East Helena and Helena indicated a suspended sulfate to total suspended particulate ratio of 0.063. For Kellogg, the assumption has been made that the ratio of suspended sulfate to total suspended particulate is the average of these values. Using the statistical analysis outlined previously, the suspended sulfate to total suspended particulate ratio for Kellogg was estimated to be 0.111 ± 0.057. Historical estimates of suspended sulfates were made by multiplying this factor by the estimate of total suspended particulate given in Table 3.1.6. The results of these calculations are also given in Table 3.1.6. During the period

1968-1970, the average suspended sulfate level was estimated to be 11.3 μg/m³. A value of 19.9 μg/m³ was estimated for the period 1964-1967. During the decade of the fifties, an average of 19.3 μg/m³ was estimated.

### Exposure Estimates for Anaconda

During the period 1961-1962, the annual total suspended particulate concentration was found to be 84.5 μg/m³. In 1971, the average suspended particulate level was 52 μg/m³. By comparing the observed total suspended particulate concentration with the particulate emitted from the Anaconda plant, a ratio of 9.1 ± 2.3 (μg/m³)/(ton/day) was determined. This ratio was multiplied by the particulate emission for Anaconda shown in Table 3.1.3 to estimate the total suspended particulate concentrations for the years 1940-1970. The results of these calculations are shown in Table 3.1.7. Estimated total suspended particulate concentrations averaged 65 μg/m³ during 1968-1970, which represented a moderate decrease from an estimated average of 69 μg/m³ during 1964-1967.

For 1965, the Montana State Department of Health reports the average annual concentration of sulfur dioxide in Anaconda was 80 μg/m³. For 1971, the corresponding value was 286 μg/m³. These values are based on the results of analysis of sulfation plates. Comparing these observed concentrations with the sulfur dioxide emissions in Table 3.1.2, a ratio of 0.343 ± 0.253 (μg/m³)/(ton/day) was obtained for the Anaconda area. In order to estimate the average sulfur dioxide concentrations during the period 1940-1970, the sulfur dioxide emissions shown in Table 3.1.2 were multiplied by this factor. The annual values of sulfur dioxide in Anaconda are estimated to be high (Table 3.1.7). For example, in the period 1968-1970, an average concentration of 177 μg/m³ was estimated. The corresponding estimated values were 193 μg/m³ for 1964-1967, 192 μg/m³ for 1960-1963, 153 μg/m³ for 1950-1959, and 203 μg/m³ for 1940-1949.

Since no data were available for suspended sulfate concentrations in the Anaconda area, an approach

Table 3.1.6. ESTIMATED ANNUAL AVERAGE CONCENTRATIONS OF SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE FOR KELLOGG, 1940-1971 (μg/m3)

| Year | Pollutant | | |
|---|---|---|---|
| | $SO_2$ | TSP | SS |
| 1971 | 411.8 | 99.5 | 10.9 |
| 1970 | 372.0 | 99.5 | 10.9 |
| 1969 | 377.7 | 105.7 | 11.6 |
| 1968 | 372.0 | 104.2 | 11.4 |
| 1967 | 347.9 | 193.4 | 21.2 |
| 1966 | 330.9 | 185.1 | 20.3 |
| 1965 | 311.0 | 175.5 | 19.7 |
| 1964 | 301.0 | 171.1 | 18.2 |
| 1963 | 288.3 | 162.8 | 17.8 |
| 1962 | 284.0 | 156.6 | 17.2 |
| 1961 | 198.8 | 119.0 | 13.1 |
| 1960 | 95.1 | 52.7 | 5.7 |
| 1959 | 232.9 | 134.2 | 14.7 |
| 1958 | 312.4 | 131.1 | 14.4 |
| 1957 | 369.2 | 151.6 | 16.6 |
| 1956 | 332.3 | 133.9 | 14.7 |
| 1955 | 311.0 | 125.6 | 13.8 |
| 1954 | 413.2 | 223.2 | 24.4 |
| 1953 | 410.4 | 223.8 | 24.5 |
| 1952 | 379.1 | 209.3 | 22.9 |
| 1951 | 367.6 | 203.7 | 22.3 |
| 1950 | 404.7 | 227.9 | 24.9 |
| 1949 | 291.1 | 162.1 | 17.7 |
| 1948 | 471.4 | 178.9 | 19.6 |
| 1947 | 309.6 | 171.1 | 18.7 |
| 1946 | 235.7 | 132.1 | 14.5 |
| 1945 | 257.0 | 130.8 | 14.3 |
| 1944 | 291.1 | 67.3 | 7.4 |
| 1943 | 312.4 | 68.2 | 7.5 |
| 1942 | 343.6 | 86.2 | 9.4 |
| 1941 | 336.5 | 84.3 | 9.2 |
| 1940 | 311.0 | 85.9 | 9.4 |

Table 3.1.7. ESTIMATED ANNUAL AVERAGE CONCENTRATIONS OF SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE FOR ANACONDA, 1940 - 1971 (μg/m3)

| Year | Pollutant | | |
|---|---|---|---|
| | $SO_2$ | TSP | SS |
| 1971 | 177.0 | 65.2 | 7.2 |
| 1970 | 217.8 | 80.6 | 8.9 |
| 1969 | 186.9 | 68.9 | 7.6 |
| 1968 | 125.9 | 46.2 | 5.1 |
| 1967 | 118.7 | 43.5 | 4.8 |
| 1966 | 231.5 | 86.1 | 9.6 |
| 1965 | 208.9 | 77.0 | 8.5 |
| 1964 | 215.7 | 70.7 | 7.8 |
| 1963 | 165.7 | 53.5 | 5.9 |
| 1962 | 195.5 | 63.4 | 7.0 |
| 1961 | 216.1 | 69.8 | 7.7 |
| 1960 | 191.4 | 61.6 | 6.8 |
| 1959 | 136.9 | 44.4 | 4.9 |
| 1958 | 188.7 | 60.7 | 6.7 |
| 1957 | 190.4 | 61.6 | 6.8 |
| 1956 | 200.3 | 64.3 | 7.1 |
| 1955 | 169.8 | 54.4 | 6.0 |
| 1954 | 123.5 | 39.9 | 4.4 |
| 1953 | 161.6 | 51.6 | 5.7 |
| 1952 | 128.6 | 41.7 | 4.6 |
| 1951 | 119.4 | 38.1 | 4.2 |
| 1950 | 113.2 | 36.2 | 4.0 |
| 1949 | 114.6 | 38.1 | 4.2 |
| 1948 | 121.1 | 39.0 | 4.3 |
| 1947 | 120.7 | 39.0 | 4.3 |
| 1946 | 121.8 | 39.0 | 4.3 |
| 1945 | 184.2 | 58.9 | 6.5 |
| 1944 | 244.9 | 78.8 | 8.7 |
| 1943 | 287.4 | 92.4 | 10.3 |
| 1942 | 292.2 | 94.2 | 10.5 |
| 1941 | 267.9 | 86.1 | 9.6 |
| 1940 | 268.6 | 86.1 | 9.6 |

identical to that used in Kellogg was used. The ratio of suspended sulfate concentration to the total suspended particulate concentration was estimated to be 0.11 ± 0.06 on the basis of the results found in East Helena, Helena, and Magna. Estimates of historical suspended sulfate concentrations for Kellogg were made by multiplying this factor by the total suspended particulate values shown in Table 3.1.7. The values of suspended sulfate for the time period 1940-1970 are also given in Table 3.1.7. For example, for 1968-1970, an average suspended sulfate level of 7.2 μg/m$^3$ was estimated. During the period 1964-1967, this level was estimated to be 7.7 μg/m$^3$.

## HUMAN EXPOSURE SUMMARY

A graphical summary of the estimated annual concentrations of total suspended particulate for Anaconda, East Helena, and Kellogg for the time period 1940-1970 is shown in Figure 3.1.6. In general, Anaconda is characterized by total suspended particulate levels that were below 75 μg/m$^3$, the National Primary Air Quality Standard for total suspended particulate, whereas the levels in both East Helena and Kellogg tended to be considerably above this value. In the decade of the forties, total suspended particulate in East Helena was estimated to have averaged 278 μg/m$^3$. The weighted average of 340 total suspended particulate samples taken at four stations in Bozeman during 1967-1968[7] was 50 μg/m$^3$. This value was used as an estimate of total suspended particulate concentration during the period 1940-1970 for Bozeman. For Helena, 13 years of data were available for the estimation of historical trends for total suspended particulate. These data indicate geometric means ranging from 41 to 75 μg/m$^3$.

The summary graph of the estimated sulfur dioxide concentrations for Anaconda, East Helena, and Kellogg is shown in Figure 3.1.7. Both Anaconda and Kellogg experienced estimated sulfur dioxide concentrations in excess of the present National Primary Air Quality Standard of 80 μg/m$^3$, whereas East Helena experienced concentrations consistently below this level. For example, during the decade of the fifties, East Helena was estimated to have had an average sulfur dioxide concentration of 39 μg/m$^3$ in contrast to an estimate of 353 μg/m$^3$ for Kellogg. Measured ambient sulfur dioxide levels in Bozeman during 1967 were 10 μg/m$^3$; in Helena, measured sulfur dioxide levels were 26 μg/m$^3$ in 1967 and 1969.[7,9] These respective values were used as an estimate of sulfur dioxide exposure for 1940-1970 for the latter two communities.

A similar summary of the estimated annual suspended sulfate concentrations for Anaconda, East Helena, and Kellogg is shown in Figure 3.1.8. In this case, during the decade of the sixties, Anaconda and East Helena have similar exposures, whereas those in Kellogg are considerably higher. The weighted average of 340 samples of suspended sulfates taken in Bozeman during 1967-1968[7] was 3.3 μg/m$^3$. A similar weighted average for 123 samples in Helena during 1966-1967[6] was 4.9 μg/m$^3$. These values were used to represent exposure for 1940-1970 in Bozeman and Helena.

A summary of the overall exposure to sulfur dioxide, total suspended particulate, and suspended sulfate for the five communities is given in Table 3.1.8. Since the estimated sulfur dioxide levels in Anaconda and Kellogg are so much higher than in the other communities, these communities are referred to as High I and High II. Bozeman, Helena, and East Helena are referred to, respectively, as Low I, Low II, and Low III.

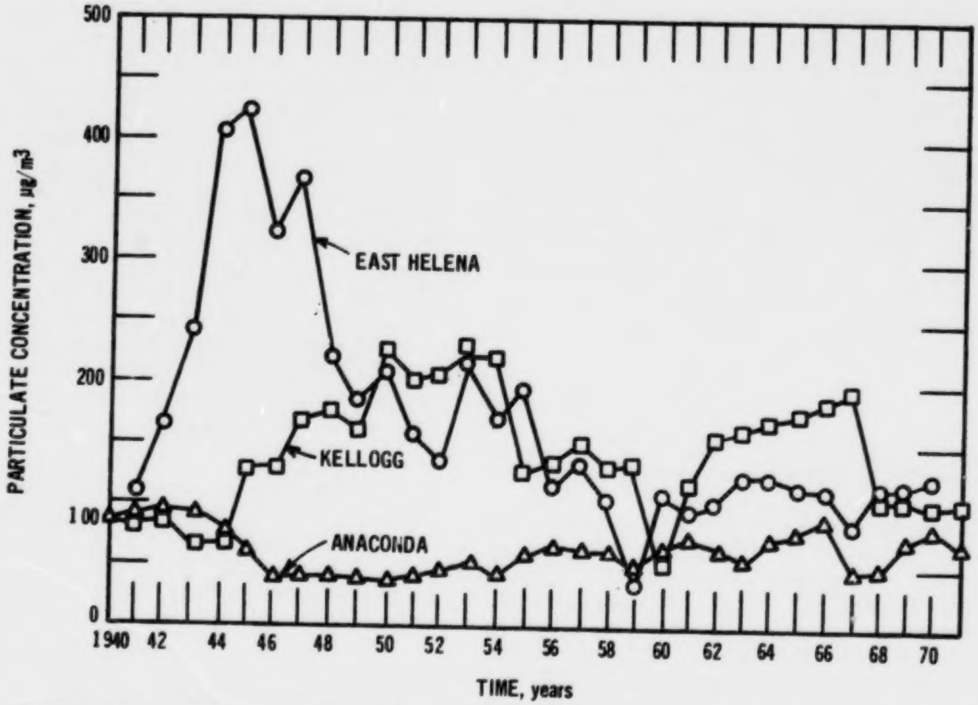

Figure 3.1.6. Estimated annual average total suspended particulate concentrations versus time for Anaconda, East Helena, and Kellogg.

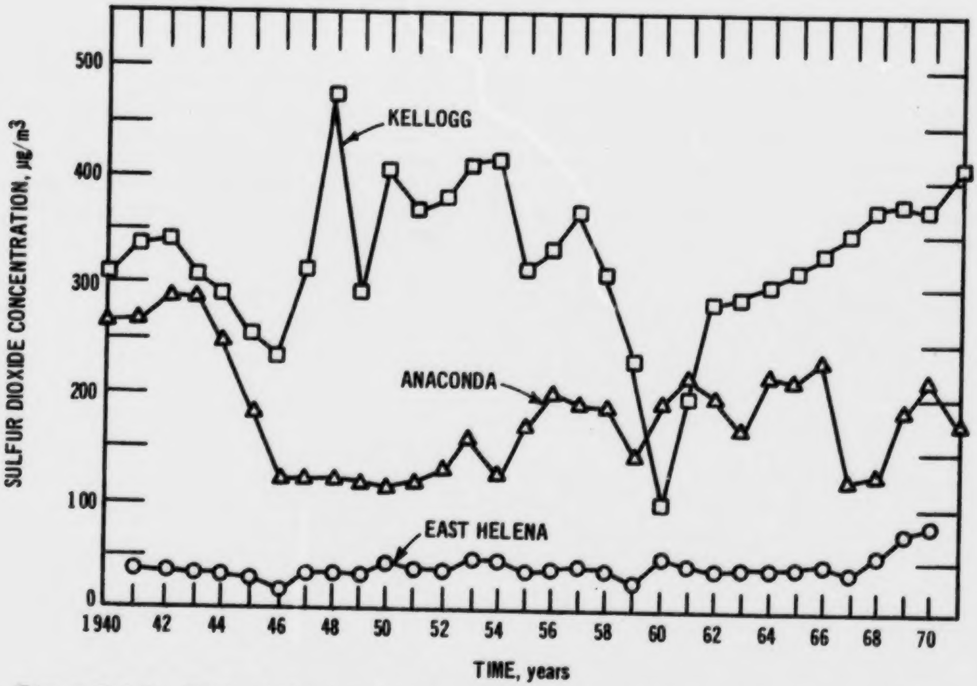

Figure 3.1.7. Estimated annual average sulfur dioxide concentrations versus time for Anaconda, East Helena, and Kellogg.

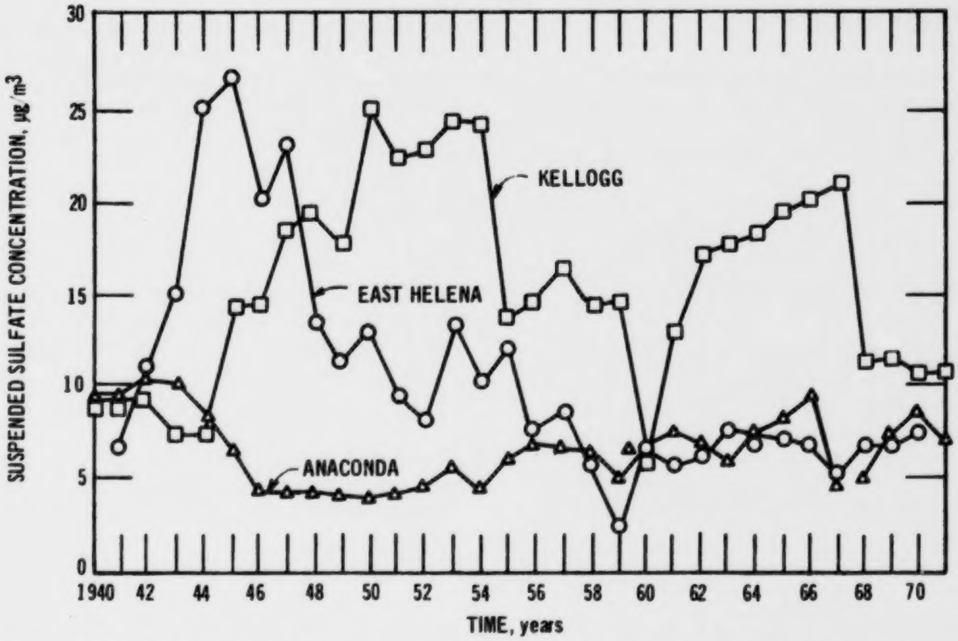

Figure 3.1.8. Estimated annual average suspended sulfate concentrations versus time for Anaconda, East Helena, and Kellogg.

**Table 3.1.8. ESTIMATED ANNUAL AVERAGE AMBIENT SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE EXPOSURES IN FIVE ROCKY MOUNTAIN COMMUNITIES, 1940-1970**

| Pollutant and community[a] | Estimated annual average concentration, µg/m³ | | | | |
|---|---|---|---|---|---|
| | 1940-1949 | 1950-1959 | 1960-1963 | 1964-1967 | 1968-1970 |
| Sulfur dioxide | | | | | |
| Low I | 10 | 10 | 10 | 10 | 10 |
| Low II | 26 | 26 | 26 | 26 | 26 |
| Low III | 34 | 39 | 40 | 38 | 67 |
| High I | 203 | 153 | 192 | 193 | 177 |
| High II | 316 | 353 | 217 | 323 | 374 |
| Total suspended particulate | | | | | |
| Low I | 50 | 50 | 50 | 50 | 50 |
| Low II | 60 | 60 | 48 | 55 | 45 |
| Low III | 270 | 146 | 106 | 107 | 115 |
| High I | 65 | 49 | 62 | 69 | 65 |
| High II | 115 | 174 | 121 | 179 | 102 |
| Suspended sulfate | | | | | |
| Low I | 3.3 | 3.3 | 3.3 | 3.3 | 3.3 |
| Low II | 4.9 | 4.9 | 4.9 | 4.9 | 4.9 |
| Low III | 17.1 | 9.2 | 6.7 | 6.8 | 7.3 |
| High I | 6.8 | 5.4 | 6.9 | 7.7 | 7.2 |
| High II | 12.8 | 19.3 | 13.5 | 19.9 | 11.3 |

[a] Low I is Bozeman; Low II, Helena; Low III, E. Helena; High I, Anaconda; and High II, Kellogg.

## ACKNOWLEDGMENTS

The authors are grateful to the Idaho and Montana State Departments of Health and to the American Smelting and Refining Company, the Anaconda Corporation, and the Bunker Hill Company (a subsidiary of Gulf Resources and Chemical Corporation) for supplying the U. S. Environmental Protection Agency with air pollutant measurements, available production and emissions data, and other helpful information regarding the history of these smelter operations.

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# 3.2 PREVALENCE OF CHRONIC RESPIRATORY DISEASE SYMPTOMS IN ADULTS: 1970 SURVEY OF FIVE ROCKY MOUNTAIN COMMUNITIES

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## INTRODUCTION

Studies in several countries have linked episodic community air pollution exposure with increased morbidity and mortality among people with chronic bronchitis.[1-12] The role of long-term exposure to lower ambient levels of sulfur dioxide in the induction and exacerbation of chronic respiratory disease is somewhat less conclusive. United States, Australian, British, Italian, and Japanese studies have related ambient sulfur dioxide and particulate exposures with chronic bronchitis prevalence and an increase in chronic respiratory symptoms in adults.[13-17] British and U.S. prospective studies of patients with chronic bronchitis have shown a positive correlation between acute symptom reporting and daily air pollution levels.[18-20]

In addition to ambient air pollution, chronic respiratory disease can result from pollution from other sources, such as occupational exposure to irritating fumes and dusts and exposure to cigarette smoke. Demographic characteristics and residential mobility also are related to risk of chronic bronchitis and less severe symptoms of chronic respiratory disease. It has not been possible to consider all of these important covariates in most previous studies. Furthermore, most studies have not been designed to distinguish between the effects of sulfur dioxide and other pollutants such as oxides of nitrogen, suspended sulfates, suspended nitrates, and total suspended particulates. This problem was summarized succinctly in the following recent comments submitted by an American industrial spokesman "...in no study was $SO_2$ the sole air pollutant.... Accordingly, none of the studies reliably isolates $SO_2$ as the causative factor which accounted for the observed differences in health effects."[21]

Western smelting communities provide an unusual opportunity to observe populations exposed to high sulfur dioxide levels without the elevated particulate levels with which they are usually accompanied. The same corporation spokesman incisively points out that, "In the comparatively open rural areas of the West where smelters are typically located, particulate levels are normally very low.... Moreover, the composition of particulate matter in western areas, where population density is low and natural gas is the man source of space heating, can be expected to be quite different from that found in the urban areas in England or the eastern United States that are dealt with in epidemiological studies."[21] The need for distinguishing the health effects of sulfur dioxide from those of other air pollutants is a compelling reason to study western smelting communities. Point source emissions from smelters usually constitute the only major source of pollution, and production and emissions data are often available for the past 20 to 30 years. Thus, one can reconstruct estimates of average ambient air concentrations of sulfur dioxide, suspended sulfates, and total suspended particulates in smelting communities for several decades.

Three smelting and two nonsmelting communities were selected for this survey of chronic respiratory disease. Available information indicated that these cities would provide a marked contrast in previous exposure to sulfur dioxide. Three specific hypotheses were proposed: first, that frequency and severity of chronic respiratory symptoms vary across areas with sulfur dioxide exposure; second, that rates of chronic bronchitis are related to length of exposure in polluted communities; third, that the combined deleterious effects of air pollution and cigarette smoking are greater than those of either factor alone.

## METHODS

### Community Selection

Five communities in the Montana-Idaho area were selected for study. In three of these communities, large smelters have been operating for over 50 years, while in the other two, there has been no heavy industry and best available information indicated no major pollution sources. In addition to the obvious pollution potential of the communities, their selection was based on routine aerometric monitoring data, collected by Montana and Idaho State health departments in conjunction with Federal agencies.[22-29] In two of the communities, daily samples from continuous monitors were available; but in others, less sophisticated monitoring techniques had been utilized, and conversions were necessary to estimate sulfur dioxide levels. Subsequently, in order to characterize previous long-term exposure, additional monitoring has been instituted and historic smelter production data calibrated to these measurements have been utilized to reconstruct earlier pollution levels.[30]

Bozeman, Montana, the southernmost city, is a farming and college community. Its 1970 population of 18,500 represented an increase of 40 percent during the previous decade. Approximately 100 miles north are Helena and East Helena. Both towns are located on a sloping valley 25 to 35 miles wide and tend to be swept by winds. Helena, the state capital, had a 1970 population of 23,000, representing a 12 percent growth since 1960. East Helena has a population of less than 1700 and has experienced an 11 percent growth since 1960. Although a lead smelter has been in operation for over 80 years in East Helena, its residents are protected to some extent by the location of the town with respect to the prevailing wind patterns. Anaconda, located 90 miles southwest of Helena in a deep valley partially surrounded by high mountains, is the site of a large copper smelter. Its 1970 population of 9800 has decreased by 19 percent since 1960. Kellogg, Idaho, 250 miles northwest of Helena, has both lead and zinc smelting operations, and is located in a narrow valley that limits ventilation. Its 1970 population of 3800 had decreased by 25 percent during the previous 10 years.

In summary, Helena and Bozeman were relatively clean nonindustrial cities, while East Helena, Anaconda, and Kellogg each had smelting operations. When compared to Anaconda and Kellogg, East Helena city residents were exposed to less of the impact of the smelter emissions.

### Data Collection

In each of the selected communities, families for study were selected through elementary school records. During November of 1970, a School and Family Health Questionnaire with explanatory letter was sent home by children to be completed by their parents and returned to the school. An example of the form used is presented elsewhere.[31] In Kellogg, Anaconda, and East Helena, all existing elementary schools were included. Only two schools chosen for their comparability were selected in each of the larger cities of Helena and Bozeman. Three of the eight schools in Anaconda were Catholic parochial. The remainder in all cities were from public school systems.

The questionnaire included a set of seven questions dealing with chronic respiratory disease symptoms (adapted for self-administration from those used by the British Medical Research Counsel).[32] Another set of questions referred to pollution exposure history, including smoking patterns, occupational exposure to irritating smoke, dust, or fumes, and residential history. In addition, age, sex, race, and education were ascertained.

### Morbidity Indices

Responses to questions regarding respiratory symptoms of shortness of breath and frequency and duration of phlegm production (from chest) and cough were used to develop a scale of severity as follows:

1. No symptoms.
2. Cough alone for less than 3 months each year.
3. Phlegm production with or without cough for less than 3 months each year.
4. Cough without phlegm production for 3 months or more each year.
5. Phlegm without cough for 3 months or more each year.
6. Cough and phlegm for 3 months or more each year.
7. Cough and phlegm for 3 months or more each year with shortness of breath.

Severity categories 6 and 7 are considered equivalent to chronic bronchitis for the purpose of this study.

Three indices were derived to summarize morbidity of various study segments: first, chronic bronchitis prevalence rates; second, mean symptom scores computed by averaging the score, or rank, on the scale of severity; and third, mean severity scores computed by averaging the symptom score only for those who reported symptoms. Through the use of these indices, one is able to gain insight into observed differences in terms of minor and severe symptoms.

### Data Analysis

Distributions of important covariates were compared across areas, as were the effects of those covariates on chronic respiratory disease. Appropriate controls or adjustments were utilized for display of the data, and a general linear model for categorical data was employed for the statistical analyses.[33] Chi Square partitioning procedures were used to test hypotheses.

## RESULTS

### Environmental Exposure

Air pollution exposure since 1940 has been reconstructed from existing aerometric measurements and available smelter production information (Table

3.2.1).[30] Sulfur dioxide exposure clearly distinguishes two of the smelter communities, Anaconda (High I) and Kellogg (High II), from the other smelter community, East Helena (Low III), and the two nonsmelter communities, Bozeman (Low I) and Helena (Low II).

This distinction is not seen in the total suspended particulate or the suspended sulfate averages. East Helena has the highest total suspended particulate average and is intermediate, more closely resembling the High communities, in suspended sulfate levels. The size of the East Helena population did not permit its analysis as a separate Intermediate pollution area; therefore, it was considered in the analyses as a Low sulfur dioxide community. Levels of sulfur dioxide, as well as total suspended particulates and suspended sulfates, have been higher on the average in the High II than in the High I community.

## Response Rates

Questionnaire response rates were similar across the five cities, ranging from 83.3 to 85.0 percent (Table 3.2.2). The small differences that were observed did not indicate a likely source of bias between the High and Low cities since the pooled response for the Low areas and for the High areas were almost equivalent. Unfortunately, nonrespondents could not be personally interviewed as has been done in more recent Community Health and Environmental Surveillance System (CHESS) studies.

## Characterization of the Study Population

Over 97 percent of the respondents were white and approximately 1 percent were either black, Indian, Mexican-American, or Oriental. The remainder did not indicate their race. Socioeconomic

Table 3.2.1. ESTIMATED ANNUAL AVERAGE AMBIENT SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE EXPOSURES IN FIVE ROCKY MOUNTAIN COMMUNITIES, 1940-1970

| Pollutant and community[a] | Estimated annual average concentration, $\mu g/m^3$ | | | | |
|---|---|---|---|---|---|
| | 1940-1949 | 1950-1959 | 1960-1963 | 1964-1967 | 1968-1970 |
| Sulfur dioxide | | | | | |
| Low I | 10 | 10 | 10 | 10 | 10 |
| Low II | 26 | 26 | 26 | 26 | 26 |
| Low III | 34 | 39 | 40 | 38 | 67 |
| High I | 203 | 153 | 192 | 193 | 177 |
| High II | 316 | 353 | 217 | 323 | 374 |
| Total suspended particulate | | | | | |
| Low I | 50 | 50 | 50 | 50 | 50 |
| Low II | 60 | 60 | 48 | 55 | 45 |
| Low III | 270 | 146 | 106 | 107 | 115 |
| High I | 65 | 49 | 62 | 69 | 65 |
| High II | 115 | 174 | 121 | 179 | 102 |
| Suspended sulfate | | | | | |
| Low I | 3.3 | 3.3 | 3.3 | 3.3 | 3.3 |
| Low II | 4.9 | 4.9 | 4.9 | 4.9 | 4.9 |
| Low III | 17.1 | 9.2 | 6.7 | 6.8 | 7.3 |
| High I | 6.8 | 5.4 | 6.9 | 7.7 | 7.2 |
| High II | 12.8 | 19.3 | 13.5 | 19.9 | 11.3 |

[a] Low I is Bozeman; Low II, Helena; Low III, E. Helena; High I, Anaconda; and High II, Kellogg.

status as indicated by education (proportion of heads of household completing high school) and crowding (percent of households with more than one room per person) reflected the industrial or nonindustrial nature of the cities (Table 3.2.2). As expected, a more educated population living in less crowded housing was found in the Low pollution communities.

Respondents were asked about exposure to coal dust, cutting oils, asbestos, mine dust, smelter fumes, or raw cotton dust through their occupation. Exclusion of those who reported prolonged exposure to these irritating dusts or fumes resulted in a loss of approximately one-third of the males from most subsequent analyses for the High pollution areas (Table 3.2.2). Parents included in all analyses are distributed in Table 3.2.3 by smoking status, sex, and age group for individual communities and for the pooled High and pooled Low pollution areas.

## Chronic Respiratory Disease Evaluation

### *Chronic Bronchitis*

Prevalence rates for chronic bronchitis are displayed by age and by educational attainment for parents in each smoking category in Table 3.2.4. A stronger age effect was seen in fathers than mothers,

Table 3.2.2. COMMUNITY CHARACTERISTICS: RESPONSE RATES, EDUCATIONAL STATUS OF FATHERS, CROWDING INDEX, AND OCCUPATIONAL EXPOSURE TO IRRITATING SMOKE, DUST, OR FUMES

| Community | Families completing questionnaire | Percent response | Education (percent of heads of household completing high school) | Crowding index (percent of households with $\leq$1.0 person/room) | Percent of fathers excluded because of occupational exposure[a] |
|---|---|---|---|---|---|
| Pooled Low | 1488 | 84.9 | 84.9 | 81.4 | 2.9 |
| Low I | 803 | 85.0 | 90.7 | 87.3 | 1.6 |
| Low II | 483 | 84.7 | 80.3 | 76.5 | 1.4 |
| Low III | 202 | 84.9 | 73.6 | 70.9 | 11.9 |
| Pooled High | 1626 | 83.5 | 60.5 | 67.9 | 35.6 |
| High I | 846 | 83.7 | 63.2 | 67.7 | 35.1 |
| High II | 780 | 83.3 | 57.5 | 71.9 | 36.1 |

[a] Three mothers from the Low exposure communities and 17 mothers from the High exposure communities were also excluded.

Table 3.2.3. NUMBER OF NONINDUSTRIALLY EXPOSED MOTHERS AND FATHERS BY SMOKING STATUS, AGE, AND COMMUNITY

|  | Nonsmokers |  |  |  | Exsmokers |  |  |  | Smokers |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  | Mothers |  | Fathers |  | Mothers |  | Fathers |  | Mothers |  | Fathers |  |
| Community | < 40 | ≥ 40 | < 40 | ≥ 40 | < 40 | ≥ 40 | < 40 | ≥ 40 | 40 | ≥ 40 | < 40 | ≥ 40 |
| Pooled Low | 448 | 293 | 197 | 204 | 137 | 87 | 188 | 227 | 342 | 176 | 253 | 228 |
| Low I | 270 | 170 | 145 | 128 | 82 | 49 | 118 | 126 | 147 | 71 | 118 | 91 |
| Low II | 115 | 92 | 36 | 51 | 37 | 29 | 37 | 76 | 122 | 83 | 87 | 100 |
| Low III | 63 | 31 | 16 | 25 | 18 | 9 | 33 | 25 | 73 | 22 | 48 | 37 |
| Pooled High | 434 | 236 | 124 | 78 | 143 | 71 | 109 | 119 | 470 | 221 | 304 | 206 |
| High I | 193 | 144 | 59 | 43 | 70 | 42 | 52 | 75 | 222 | 154 | 132 | 118 |
| High II | 241 | 92 | 65 | 35 | 73 | 29 | 57 | 44 | 248 | 67 | 172 | 88 |
| Total | 882 | 529 | 321 | 282 | 280 | 158 | 297 | 346 | 812 | 397 | 557 | 434 |

while the educational effect was inconsistent. A marked contrast between smokers and nonsmokers was apparent, with exsmokers more closely resembling nonsmokers. Smoking fathers had more bronchitis than smoking mothers in all age groups. For nonsmokers and exsmokers, this male excess was only seen in the older age groups.

Smoking- and sex-specific rates for each community, given in Table 3.2.5, are quite variable due to the relatively small numbers of cases in most categories. When pooled High and pooled Low communities were compared, bronchitis prevalence was higher within the High exposure community in five of the six smoking- and sex-specific categories. Likewise, sex- and age-adjusted rates within the pooled High exposure population showed excess bronchitis in each smoking category. Differences were statistically significant only in the combined nonsmoking and exsmoking population (Table 3.2.6); however, there were approximately equal differences between High and Low exposure areas in smokers of both sexes. Sex-specific smoking intensity comparisons between the High and Low exposure communities are shown in Table 3.2.7. The similarity between High and Low exposure communities and the absence of a pattern in the slight differences that occur indicates that the observed area differences among smokers cannot be explained by smoking intensity.

**Table 3.2.4. SMOKING- AND SEX-SPECIFIC PREVALENCE RATES (percent) FOR CHRONIC BRONCHITIS BY EDUCATION AND AGE[a]**

| Category | Nonsmokers | | Exsmokers | | Smokers | |
|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers |
| Education: | | | | | | |
| <High school | 2.06 | 3.23 | 5.83 | 4.81 | 14.50 | 21.18 |
| High school | 2.20 | 3.66 | 1.43 | 3.49 | 11.75 | 15.70 |
| >High school | 1.36 | 1.95 | 2.51 | 2.13 | 10.85 | 19.46 |
| Age: | | | | | | |
| ≤29 | 1.09 | 0.00 | 2.63 | 0.00 | 13.07 | 14.55 |
| 30-39 | 1.31 | 0.68 | 3.86 | 2.72 | 11.17 | 15.59 |
| 40-49 | 2.63 | 4.10 | 2.38 | 3.24 | 14.95 | 21.00 |
| ≥50 | 2.61 | 6.25 | 0.00 | 5.06 | 7.69 | 28.41 |

[a] Chronic bronchitis rates are equivalent to crude rates for symptom severities 6 and 7.

**Table 3.2.5. PREVALENCE OF CHRONIC BRONCHITIS IN NONINDUSTRIALLY EXPOSED PARENTS: INDIVIDUAL AND POOLED COMMUNITY RATES (percent) BY SEX AND SMOKING STATUS**

| Community | Nonsmokers | | Exsmokers | | Smokers | | Sex- and age-adjusted rates | | |
|---|---|---|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers | Non-smokers | Ex-smokers | Smokers |
| Pooled Low | 1.08 | 1.25 | 3.12 | 1.45 | 11.78 | 17.05 | 1.08 | 2.46 | 14.00 |
| Low I | 1.36 | 1.10 | 3.05 | 0.00 | 8.72 | 12.44 | | | |
| Low II | 0.48 | 0.00 | 4.55 | 5.31 | 14.15 | 20.86 | | | |
| Low III | 1.06 | 4.88 | 0.00 | 0.00 | 13.68 | 20.00 | | | |
| Pooled High | 2.54 | 3.47 | 2.80 | 4.82 | 12.88 | 18.63 | 3.12 | 3.56 | 15.72 |
| High I | 3.56 | 4.90 | 1.79 | 4.72 | 13.83 | 18.40 | | | |
| High II | 1.50 | 2.00 | 3.92 | 4.95 | 11.75 | 18.85 | | | |

## Table 3.2.6. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS IN SMOKERS AND NONSMOKERS, CHRONIC BRONCHITIS

| Factor | Degrees of freedom | Smokers | | Nonsmokers[a] | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability (p) | $X^2$ | Probability (p) |
| Sex | 1 | 14.70 | <0.0003 | 1.12 | <0.3 |
| Education | 1 | 3.15 | <0.07 | 0.48 | <0.6 |
| Age | 1 | 8.61 | <0.004 | 10.10 | <0.002 |
| Exposure | 1 | 0.66 | <0.5 | 8.73 | <0.005 |
| Fit of model | 11 | 14.19 | <0.2 | 13.85 | <0.2 |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a larger sample size.

## Table 3.2.7. SMOKING INTENSITY AMONG SMOKERS IN POOLED HIGH AND LOW EXPOSURE COMMUNITIES

| Cigarettes smoked per day | Percent smoking | | | |
|---|---|---|---|---|
| | Fathers | | Mothers | |
| | Low | High | Low | High |
| 1 to 5 | 9.3 | 8.8 | 12.8 | 9.1 |
| 6 to 15 | 15.0 | 16.7 | 18.4 | 19.0 |
| 16 to 25 | 48.5 | 52.9 | 51.4 | 52.7 |
| > 25 | 27.3 | 21.6 | 17.4 | 19.2 |

### *Respiratory Symptom and Severity Scores*

In each of the six smoking- and sex-specific comparisons (Table 3.2.8), High exposure communities had higher symptom scores. As indicated in Table 3.2.9, area differences among nonsmokers were highly significant ($p < 0.002$), and area differences among smokers approached significance ($p < 0.07$). Again, the pattern of differences was very consistent. Symptom scores were also higher for males and for smokers within each pooled exposure group.

## Table 3.2.8. MEAN SYMPTOM SCORES IN NONINDUSTRIALLY EXPOSED PARENTS: INDIVIDUAL AND POOLED COMMUNITY AVERAGES BY SEX AND SMOKING STATUS

| Community | Nonsmokers | | Exsmokers | | Smokers | | Sex- and age-adjusted rates | | |
|---|---|---|---|---|---|---|---|---|---|
| | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers | Non-smokers | Ex-smokers | Smokers |
| Pooled Low | 1.23 | 1.33 | 1.30 | 1.41 | 2.20 | 2.65 | 1.27 | 1.35 | 2.39 |
| Low I | 1.21 | 1.27 | 1.20 | 1.32 | 1.98 | 2.53 | | | |
| Low II | 1.20 | 1.32 | 1.49 | 1.68 | 2.31 | 2.72 | | | |
| Low III | 1.34 | 1.71 | 1.23 | 1.27 | 2.47 | 2.75 | | | |
| Pooled High | 1.29 | 1.38 | 1.45 | 1.58 | 2.34 | 2.72 | 1.34 | 1.49 | 2.52 |
| High I | 1.31 | 1.46 | 1.44 | 1.58 | 2.42 | 2.87 | | | |
| High II | 1.28 | 1.30 | 1.46 | 1.58 | 2.23 | 2.58 | | | |

## Table 3.2.9. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS IN SMOKERS AND NONSMOKERS, MEAN RESPIRATORY SYMPTOM SCORES

| Factor | Degrees of freedom | Smokers | | Nonsmokers[a] | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability (p) | $X^2$ | Probability (p) |
| Sex | 1 | 32.39 | <0.0001 | 20.60 | <0.0001 |
| Education | 1 | 3.00 | <0.08 | 0.54 | <0.5 |
| Age | 1 | 3.02 | <0.08 | 6.84 | <0.009 |
| Exposure | 1 | 3.32 | <0.07 | 10.32 | <0.002 |
| Fit of model | 11 | 17.53 | <0.09 | 11.58 | <0.04 |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a larger sample size.

Mean severity scores among parents who reported some chronic respiratory symptoms did not differ between High and Low exposure communities or between sexes (Tables 3.2.10 and 3.2.11). Thus, there was no evidence for overreporting or exaggeration of minor symptoms among symptom-positive respondents from High exposure communities. Lower severity scores in High exposure communities would be anticipated if overreporting of minor symptoms occurred. Exsmokers and cigarette smokers had higher severity scores than nonsmokers. These results suggest that the severity of chronic respiratory symptoms, once present, did not differ between exposure groups but did differ between smokers and nonsmokers.

### *Length of Residence*

In order to assess the importance of duration of exposure to community air pollution, chronic bronchitis rates were computed according to the length of residence in High or Low pollution areas. Rates for smokers and current nonsmokers were computed. Within each residence duration category, the ratio of bronchitis in High exposure communities to bronchitis in Low exposure communities was calculated (Figure 3.2.1). Absolute bronchitis rates in Low exposure communities are given in Table 3.2.12. Recent immigrants (duration of residence of less than 2 years) who were nonsmokers or exsmokers had more disease in all areas than residents of 2 or more

Table 3.2.10. MEAN SYMPTOM SEVERITY SCORES FOR NONINDUSTRIALLY EXPOSED PARENTS WHO REPORTED SYMPTOMS: INDIVIDUAL AND POOLED COMMUNITY AVERAGES BY SEX AND SMOKING STATUS

| | Nonsmokers | | Exsmokers | | Smokers | | Sex- and age-adjusted rates | | |
|---|---|---|---|---|---|---|---|---|---|
| Community | Mothers | Fathers | Mothers | Fathers | Mothers | Fathers | Non-smokers | Ex-smokers | Smokers |
| Pooled Low | 4.27 | 4.05 | 4.73 | 4.11 | 4.23 | 4.52 | 4.13 | 4.47 | 4.38 |
| Low I | 4.24 | 4.08 | 5.33 | 3.89 | 4.29 | 4.44 | | | |
| Low II | 4.38 | 4.11 | 5.00 | 4.50 | 4.20 | 4.58 | | | |
| Low III | 4.20 | 3.90 | 3.00 | 3.67 | 4.18 | 4.55 | | | |
| Pooled High | 3.98 | 4.50 | 4.31 | 4.38 | 4.33 | 4.48 | 4.24 | 4.29 | 4.41 |
| High I | 4.39 | 4.61 | 4.07 | 4.36 | 4.40 | 4.52 | | | |
| High II | 3.62 | 4.33 | 4.62 | 4.41 | 4.24 | 4.43 | | | |

Table 3.2.11. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS IN SMOKERS AND NONSMOKERS, SEVERITY SCORES FOR THOSE WITH SYMPTOMS

| Factor | Degrees of freedom | Smokers | | Nonsmokers[a] | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability (p) | $X^2$ | Probability (p) |
| Sex | 1 | 3.53 | <0.05 | 0.11 | <0.8 |
| Education | 1 | 0.20 | <0.8 | 2.11 | <0.14 |
| Age | 1 | 9.72 | <0.002 | 3.59 | <0.055 |
| Exposure | 1 | 0.06 | <0.09 | 1.51 | <0.2 |
| Fit of model | 11 | 6.65 | <0.82 | 20.35 | <0.04 |

[a] Exsmokers and lifetime nonsmokers were combined for this analysis to obtain a larger sample size.

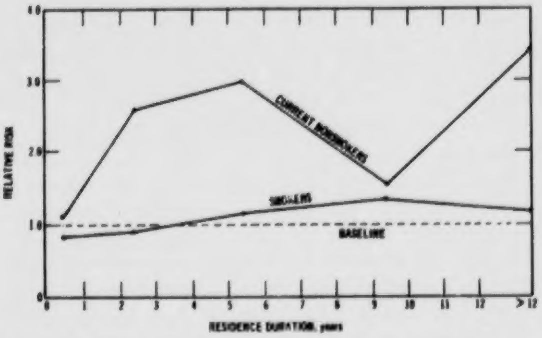

Figure 3.2.1. Risk of chronic bronchitis in High exposure communities relative to baseline experience of Low exposure communities.

Table 3.2.12. CHRONIC BRONCHITIS ATTACK RATES (percent) FOR SMOKERS AND CURRENT NONSMOKERS IN LOW EXPOSURE COMMUNITIES BY DURATION OF RESIDENCE[a]

| Smoking status | Residence duration, years | | | | |
|---|---|---|---|---|---|
| | ≤1 | >1 to ≤3 | >3 to ≤7 | >7 to ≤12 | >12 |
| Current nonsmokers | 2.4 | 11.0 | 0.7 | 1.5 | 1.8 |
| Smokers | 16.1 | 17.0 | 13.5 | 9.9 | 14.4 |

[a] Baseline rates for Figure 3.2.1.

years duration. In High exposure communities, smoking and nonsmoking long-time residents (more than 4 years) had more disease than residents of intermediate duration. Residence duration-specific bronchitis ratios were greater than unity among nonsmoking and smoking residents of intermediate and long duration in High exposure communities. Of particular importance was the excess bronchitis in nonsmoking adults of 2 to 3 years residence duration in High exposure communities. These data suggested a relatively recent effect of exposure on chronic disease prevalence. A trend towards increasing excess bronchitis with longer residence in High exposure communities was also apparent among nonsmokers and smokers. Excess bronchitis among smokers in High exposure areas was relatively less than among nonsmokers, reflecting the larger effect of cigarette smoking, compared with pollutant exposure, on bronchitis prevalence.

### *Relative Importance of Community Air Pollution and Cigarette Smoking*

In order to distinguish the relative effects of cigarette smoking and air pollution, chronic bronchitis rates for those who had been exposed to each of these factors alone were compared with rates for those presumably exposed to neither factor (Table 3.2.13). Prevalence rates were computed for nonsmokers, exsmokers, and smokers residing in polluted and in clean areas. Bronchitis rates of nonsmokers from clean areas were used as a baseline.

Table 3.2.13. RELATIVE IMPORTANCE OF CIGARETTE SMOKING AND AMBIENT AIR QUALITY AS DETERMINED BY COMPARISON OF EXCESS PREVALENCE OF CHRONIC BRONCHITIS

| Sex and smoking status | Community air quality (pooled) | Excess prevalence[a] | Additive model for smoking and pollution | |
|---|---|---|---|---|
| | | | Expected | Observed/ expected |
| **Female** | | | | |
| Lifetime nonsmoker | Clean | 0.00<br>(1.08) | – | – |
| | Dirty | 1.46 | – | – |
| Exsmoker | Clean | 2.04 | – | – |
| | Dirty | 1.72 | 3.50 | 0.49 |
| Smoker | Clean | 10.70 | – | – |
| | Dirty | 11.80 | 12.16 | 0.97 |
| **Male** | | | | |
| Lifetime nonsmoker | Clean | 0.00<br>(1.25) | – | – |
| | Dirty | 2.22 | – | – |
| Exsmoker | Clean | 0.20 | – | – |
| | Dirty | 3.57 | 2.42 | 1.48 |
| Smoker | Clean | 15.80 | – | – |
| | Dirty | 17.38 | 18.02 | 0.96 |

[a] Base rates in parentheses. Excess prevalence = smoking- and community-specific prevalence rate minus base rate.

The isolated effect of pollution was computed by the excess prevalence among nonsmokers of High versus Low exposure communities. This difference was 1.46 for females and 2.22 for males. The isolated effect of smoking was computed as the difference in prevalence between smokers and nonsmokers in Low exposure communities. These differences were 10.70 for female smokers and 15.80 for male smokers. Thus, among both females and males, the effect of air pollution on bronchitis prevalence was 14 percent of the effect of smoking. The effect of current air pollution approached that of past cigarette smoking (in exsmokers) on bronchitis prevalence among females. In males, current air pollution had a stronger effect than past cigarette smoking.

If the effects of air pollution and smoking were additive, excess bronchitis among smokers in Low exposure communities added to excess bronchitis among nonsmokers in High exposure communities should equal the excess bronchitis among smokers in High exposure communities. In each case, excess bronchitis must be derived with baseline rates of nonsmokers in Low exposure communities. When these calculations were made, 97 percent of the excess bronchitis expected in High exposure areas among smoking females and 96 percent among smoking males could be accounted for by the simple additive model. However, the additive model underestimated the combined effect of exsmoking and pollution among females, and overestimated this combined effect among males.

### *Occupational Exposure*

Smoking- and age-specific chronic bronchitis prevalence rates, mean symptom scores, and severity scores for males with and without occupational exposure in High exposure communities were computed (Table 3.2.14). For this analysis, occupational exposure was defined as: exposure on the job for at least 5 years to either coal dust, cutting oils, asbestos, mine dust, smelter fumes, or raw cotton dust. There were 478 males with occupational exposure and 940 without occupational exposure. Consistently higher prevalence rates and symptom scores were observed in occupationally exposed males of each smoking-age category. Severity scores were higher in occupationally exposed smokers but lower in nonsmokers with occupational exposure. Results indicate that bronchitis morbidity is increased in smokers and nonsmokers by community and occupational exposures, and that exclusion of occupationally exposed males from previous analyses yielded a conservative estimate of the bronchitis morbidity effect of community air pollution.

Utilizing the model previously used to test for additive effects of cigarette smoking and community exposure, the effects of occupational exposure were evaluated. Since occupational exposure in the Low pollution communities was quite rare, its effect was estimated as the difference between the prevalence in occupationally exposed and nonoccupationally exposed nonsmokers in the High pollution communities. This difference was added to the previously calculated excess prevalence for smoking status and community exposure to derive an expected excess risk for those individuals who had experienced exposure from all three sources (Table 3.2.15). The additive model predicted 82 and 88 percent of the excess risks, again indicating that the effects of occupational exposure, superimposed on community exposure and cigarette smoking, appear to be additive.

**Table 3.2.14. BRONCHITIS PREVALENCE, SYMPTOM SCORES, AND SEVERITY SCORES BY AGE IN MALES WITH AND WITHOUT OCCUPATIONAL EXPOSURES IN POOLED HIGH EXPOSURE COMMUNITIES**

| Smoking category | Occupational dust exposure | Prevalence, percent |  | Symptom score |  | Severity score |  |
|---|---|---|---|---|---|---|---|
|  |  | <40 | ≥40 | <40 | ≥40 | <40 | ≥40 |
| Nonsmokers and exsmokers | Yes | 2.2 | 7.0 | 1.62 | 1.90 | 3.80 | 4.68 |
|  | No | 2.1 | 6.6 | 1.33 | 1.66 | 4.39 | 4.79 |
| Smokers | Yes | 17.4 | 27.3 | 2.89 | 3.40 | 4.49 | 4.87 |
|  | No | 13.8 | 25.7 | 2.50 | 3.05 | 4.29 | 4.71 |

Table 3.2.15. RELATIVE IMPORTANCE OF OCCUPATIONAL EXPOSURE, CIGARETTE SMOKING, AND AMBIENT AIR QUALITY AS DETERMINED BY COMPARISON OF EXCESS RISK OF CHRONIC BRONCHITIS

| Smoking status | Community air quality (pooled) | Occupational exposure | Excess prevalence of chronic bronchitis | Additive model for smoking, occupational exposure, and pollution | |
|---|---|---|---|---|---|
| | | | | Expected | Observed/ expected |
| Lifetime nonsmoker | Clean | Unexposed | 0.00<br>(1.25)[a] | | |
| | Dirty | Unexposed | 2.22 | | |
| | Dirty | Exposed | 4.43 | | |
| Exsmoker | Clean | Unexposed | 0.20 | | |
| | Dirty | Unexposed | 3.57 | | |
| | Dirty | Exposed | 3.78 | 4.63 | 0.82 |
| Smoker | Clean | Unexposed | 15.80 | | |
| | Dirty | Unexposed | 17.11 | | |
| | Dirty | Exposed | 17.78 | 20.23 | 0.88 |

[a] Base rate in parentheses.

## DISCUSSION

In two Rocky Mountain smelter communities exposed to elevated levels of atmospheric sulfur dioxide and suspended sulfates, but not to total suspended particulates, the prevalence of chronic bronchitis and overall mean respiratory symptom scores were significantly increased in nonsmokers and exsmokers by comparison with three Low exposure communities in the same region. Excess bronchitis morbidity occurred among nonsmokers, exsmokers, and current smokers of both sexes in the High exposure smelter communities.

Three hypotheses were proposed and supported by this study: First, chronic bronchitis morbidity was related to sulfur dioxide exposure. Second, the excess of chronic bronchitis tended to increase with longer duration of residence in High exposure communities. Third, the combined deleterious effects of air pollution and cigarette smoking were greater than those of either factor alone.

The distribution of certain important covariates of bronchitis prevalence differed between study communities due to differences in principal occupations among areas. A substantial portion of fathers in High exposure communities were excluded from most analyses because of occupational dust and fume exposure, the effects of which could not be isolated from those of community exposure. Further, in High exposure communities, educational attainment was below that of Low exposure areas. However, no consistent or statistically significant association between educational attainment and bronchitis prevalence was found in the five study communities. Differences in age distribution occurred but did not correspond to exposure contrasts between communities. Bronchitis prevalence increased with age among males in all smoking categories, but not among females. All bronchitis morbidity indices were therefore age adjusted. This adjustment compensated in part for area differences in educational achievement, since the more educated communities were generally younger. As anticipated, a strong and statistically significant male excess in bronchitis morbidity was present with each smoking category. Analyses of age-adjusted bronchitis rates were performed for sex- and smoking-specific groups or were adjusted for both age and sex within each smoking category. Smoking categories in High and Low communities were very similar with respect to the distribution of smoking intensity. Thus, differences between smokers in High and Low communities could not be attributed to area differences in smoking intensity.

The mean respiratory symptom score, based on a seven-point ordinal scale of chronic respiratory symptom reporting, proved to be a useful index. The symptom score avoided making a simple dichotomy of symptom responses into disease or no disease. Early or less prolonged chronic respiratory symptoms were given greater weight than complete absence of symptoms. The symptom score showed a more consistent response gradient between communities within each smoking-sex category than was true for bronchitis prevalence rates (compare Tables 3.2.5 and 3.2.8). The consistency may be accounted for by the fact that the spectrum of morbidity is broader with the symptom score and that when sample sizes were small; the symptom score was less variable in the presence of a low morbidity prevalence. The symptom score strongly confirmed results indicated by prevalence rates. Symptom scores have the disadvantage that relative and excess rates cannot be computed and comparison with past studies cannot be made.

Severity scores in the population reporting any respiratory symptoms showed that disease, though present in excess, was not more severe in the High exposure community. In addition, the severity score was useful in eliminating the possibility that overreporting bias could explain observed community differences in morbidity. The severity score gave no evidence that less severe or minor respiratory symptoms were exaggerated or overreported in any of the sex- and smoking-specific categories within High exposure communities. If less severe symptoms had been overreported, lower severity scores would have been anticipated from High exposure areas. This clearly was not so.

Analysis of bronchitis morbidity by duration of residence proved to be very important. The association of excess disease with recent change of residence[34-39] was confirmed in this study and demonstrated the need to analyze separately those who moved within the past year. In High exposure areas, a U-shaped disease distribution was observed over a residence duration scale. High morbidity occurred in most recent immigrants and in long-time residents. When most recent immigrants were excluded, the risk of bronchitis in High relative to Low exposure communities increased with longer residence time. This pattern was more consistent for smokers than nonsmokers. However, nonsmokers present in High exposure areas for only 2 to 3 years had 2.6-fold more bronchitis than similar residents of Low exposure areas. Smokers of 2 to 3 years residence duration did not as yet have bronchitis in excess by comparison with smokers in Low exposure areas. These observations suggested that nonsmokers may respond more rapidly to a new but polluted environment. Such effects might be anticipated, given the relatively greater chronic insult of smoking compared with air pollution. An additional contribution to the relative risk below unity among smokers who resided in High exposure areas less than 4 years was the possibility that smokers with respiratory symptoms would be disinclined to move into a polluted environment.

A further important conclusion for air quality standards could be derived from the residence duration analysis. To relate chronic disease to environmental exposure, environmental standards require intelligence concerning length of exposure as well as concentration. Oftentimes, only current air quality data are available. Assumptions are made that excess chronic disease, requiring cumulative exposure, was caused by long-term exposures similar to current levels. This assumption cannot usually be supported, especially in recent years when vigorous efforts at air pollution abatement have been underway. Attributing chronic disease to recent exposure could result in unduly restrictive standards if the disease resulted from past high exposures. This challenges the environmental health scientist to define the time-weighted concentration likely to cause excess chronic disease, a task that is formidable and usually beyond current knowledge. However, documentation of excess bronchitis in 2- to 3-year residents of High exposure communities provides evidence that current levels, which can be readily quantified, can be associated with excess disease. This association is valid if the 2- to 3-year residents moved from a low exposure area, an assumption that unfortunately could not be validated because data on location of prior residence were not obtained. Furthermore, the pattern of increasing excess illness with longer length of residence in High exposure areas is consistent with the above assumptions. Therefore, the finding of excess bronchitis in 2- to 3-year residents who were nonsmokers provides evidence that exposure to sulfur dioxide in the range of 177 to 374 $\mu g/m^3$ and suspended sulfates of 7.2 to 19.9 $\mu g/m^3$ for 2 to 3 years can result in excess chronic bronchitis; and for smokers, the corresponding period is 4 to 7 years. These exposures occurred in the presence of only moderately high total suspended particulates, 65 to 179 $\mu g/m^3$. Since High exposure areas were nonferrous smelter communities, the possibility that metallic particulates potentiated observed effects is distinctly present, especially in view of experimental findings that metallic sulfates have greater adverse effects on the respiratory tract than does sulfur dioxide.[40,41]

Comparisons of prevalence rates for bronchitis among nonsmokers and smokers in Low and High exposure communities revealed a smoking effect about 7 times greater than air pollution in this population. Simple addition of the effects of these two variables explained remarkably well the observed excess bronchitis among smokers living in High exposure communities. That is, excess bronchitis due to community air pollution was of the same magnitude in smokers and nonsmokers.

Occupationally exposed males had excess bronchitis over and above that attributable to community air pollution. Again, an additive model using community air pollution, occupational exposure, and cigarette smoking as factors was sufficient to reasonably explain the excess bronchitis among smoking, occupationally exposed males in High exposure communities. To protect the health of workers living in High exposure communities, occupational health standards should be coupled with ambient air quality standards. High community exposure forces industry to hire more vulnerable populations who can ill afford any added pollutant exposure. Thus, when starting with a higher baseline of illness, protection of employee health requires simultaneous reduction of community and occupational hazards.

One of the Low exposure communities (Low III) was a smelter town. Residential areas of this town were situated upwind of the smelter in a valley where prevailing winds minimized the entire impact of the smelter emissions. However, total suspended particulates (106 to 270 $\mu g/m^3$) and suspended sulfate concentrations (6.7 to 17.1 $\mu g/m^3$) were higher than levels in the other two Low exposure communities (45 to 60 $\mu g/m^3$ for total suspended particulate; 2.9 to 3.8 $\mu g/m^3$ for suspended sulfates). Sulfur dioxide was low in all Low exposure communities (10 to 67 $\mu g/m^3$). Increased exposure to particulates in the Low exposure smelter community (Low III) appeared to exert an adverse effect on bronchitis morbidity, as evidenced by higher mean respiratory symptom scores (Table 3.2.8) among nonsmokers and smokers of both sexes when compared with comparable groups in the other two Low exposure areas. The total number of respondents from this smelter community was too small to draw firm conclusions from these findings. Small sample sizes were reflected in the instability of bronchitis prevalence rates within smoking- and sex-specific groups for each community (Table 3.2.5). Inclusion of this smelter community in the pooled Low exposure group tended to raise baseline morbidity rates and thus to diminish the magnitude of excess bronchitis found in High exposure communities.

## SUMMARY

Chronic bronchitis prevalence and symptom scores in nonoccupationally exposed adult residents of two High exposure Rocky Mountain smelter communities significantly exceeded morbidity rates found in residents of three Low exposure communities. Age-adjusted excess bronchitis was observed within sex and smoking categories. Nonsmokers in High exposure communities had excess bronchitis prevalence rates 2.4 to 2.8 times those of nonsmokers in Low exposure areas. The effect of community exposure was additive to that of cigarette smoking, which exerted an effect on chronic bronchitis approximately 7 times stronger than that of air pollution. Symptom scores confirmed area differences shown from bronchitis prevalence rates. Though more prevalent, bronchitis was not more severe in High exposure communities. Comparison of residence duration-specific rates demonstrated that prolonged residence in High exposure areas magnified the excess bronchitis attributable to pollutant exposure. Occupational exposure further increased observed bronchitis morbidity. Excess bronchitis occurred with 2- to 3-year exposure to sulfur dioxide concentrations of 177 to 374 $\mu g/m^3$ and suspended sulfate concentrations of 7.2 to 19.9 $\mu g/m^3$ in the presence of low total suspended particulates. Metallic sulfates may well have accounted for the findings of excess bronchitis.

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# 3.3 FREQUENCY OF ACUTE LOWER RESPIRATORY DISEASE IN CHILDREN: RETROSPECTIVE SURVEY OF FIVE ROCKY MOUNTAIN COMMUNITIES, 1967-1970

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## INTRODUCTION

The purpose of this study was to determine if long-term or short-term exposures to air pollutants will aggravate or induce acute respiratory illnesses in children. Acute respiratory diseases are the most common cause of illness in children, and the lower respiratory component may very well be a portent of chronic respiratory disease in adults.[1-3] Unlike adults, elementary school children are generally not subjected to occupational pollution exposures or self-pollution by cigarette smoking. Children are also less likely to have experienced a variety of complicated long-term ambient air pollution exposures related to residential mobility. Hence, they are an excellent group in which to isolate the adverse health effects induced by community air pollution.

Several foreign studies have implicated sulfur dioxide and particulates in ambient air as a cause of increased respiratory symptoms and illnesses in children. In England, different studies of children have shown an increased frequency and severity of acute upper and lower respiratory morbidity and a decrease in pulmonary function among exposed children.[4-6] In Japan, Toyama found that 10- and 11-year old school children living in a highly polluted community had a higher frequency of nonproductive cough, upper respiratory tract irritation, and increased mucous secretion than comparable children in a less polluted area.[7] Manzhenko reported similar findings in exposed Russian children as well as a high prevalence of abnormal pulmonary X-ray findings.[8] None of these studies were designed to distinguish the effects of individual air pollutants such as sulfur dioxide, total suspended particulates, and suspended sulfates from the more complex urban mixtures. In addition, the lifetime pollution exposure doses of the children could not be calculated and the duration of exposure necessary to induce effects was not usually described. However, two studies of families living near a point source of nitrogen dioxide were able to link significant increases in respiratory illness to nitrogen dioxide exposures lasting 3 or more years.[9,10]

Respiratory morbidity in children is influenced by age, sex, and socioeconomic status. Duration of residence may be an important determinant of disease because of community pollution exposure and because individuals moving into a new community may initially suffer higher rates of illness.[11] Children with a history of asthma do have a higher frequency of lower respiratory illness than do nonasthmatics.[6] These personal covariates must be isolated or adjusted in studies that quantify the relationship between ambient air pollution and acute respiratory illness.

High levels of sulfur dioxide often occur in western smelting communities where the total suspended particulate levels are often quite low. Many of these towns are small and have relatively little other pollution from automobiles or fossil fuels. Production and emissions data for the past years are usually available. Since smelters are point sources of pollutants, one can model previous community exposures to sulfur dioxide and particulates for several decades. We describe here a retrospective survey of the frequency of acute lower respiratory illnesses among elementary school children in five Rocky Mountain communities.

## MATERIALS AND METHODS

### Community Selection

Five communities were selected on the basis of air quality data published by the States of Idaho and Montana and advice from health department officials in both states.[12-16] This study was a sequel to the Helena Valley Area Environmental Report.[17] Communities were ranked Low or High on the basis of their estimated exposure to sulfur dioxide during the period covered by the study and for the prior decade. Bozeman, the Low I community, is a prosperous, growing, farming and college community of 18,500 located about 100 miles southeast of Helena. Helena, the Low II community, is the state capitol of Montana, has a population of 23,000 and is located in a broad, usually well ventilated valley. East Helena, the Low III community, is a small town of 1700 just 3 miles from Helena and has been the site of a lead smelter since 1888, a zinc plant since 1927, and a paint pigment plant since 1955. Virtually all East Helena residents do their major shopping in Helena, and some of them work in Helena. Anaconda, the High I community, with a population of about 9800, is located about 90 miles southwest of Helena and has been the site of a large copper smelter since the end of the last century. Kellogg, the High II community, with a population of 3800, has been the site of a large lead smelter for over 75 years and is in Shoshone County, Idaho, about 252 miles northwest of Helena. It is located at the bottom of a narrow valley in the world famous Couer d'Alene mining district in the Idaho panhandle. Both Anaconda and Kellogg reported population losses in the 1970 census.

### Collection of Health and Demographic Data

Elementary school children (grades 1 to 6) in each city were asked in November 1970 to take home an explanatory letter and questionnaire to their parents. All elementary schools in Kellogg, East Helena, and Anaconda were included in the survey. Socioeconomically similar schools in the larger cities, Helena and Bozeman, were selected after consultation with the Superintendent of Schools and school principals. The frequency of lower respiratory disease in children was ascertained by means of a School and Family Health questionnaire,[18] which was completed by the mother in each family and returned to the school. The mother was instructed to give respiratory disease information on all children in the family 12 years of age or younger.

The questionnaire inquired about the frequency of treatment by a physician for pneumonia, croup, or bronchitis (including bronchiolitis or deep chest infections other than pneumonia or croup) during the 3-year period beginning in September 1967. Hence, all rates used in the analyses (except in the three figures) are 3-year rates. Other information ascertained included hospitalizations for lower respiratory illnesses, name of children's physician, history of asthma, length of residence in the community, socioeconomic status measured by education of head of household, parents' smoking status, and family census. Physician records were reviewed to validate parental reporting for approximately a 15 percent sample of questionnaires. The sample included an equal number of children reported sick and well. Additionally, a sample of 18 physicians in the study areas were queried about their definition of the bronchitis syndrome.

### Assessing Air Pollution Exposures

Annual estimated averages of sulfur dioxide, total suspended particulates, and suspended sulfates were derived from production figures and emissions estimates. A detailed report of these procedures is given elsewhere.[19]

### Hypotheses Testing

The hypotheses tested were: (1) that the frequency of physician treatment for any lower respiratory illness, croup, bronchitis, and pneumonia, as well as the frequency of hospitalization for these illnesses, adjusted for appropriate covariates, would correspond to pollutant exposures, (2) that appropriately adjusted acute lower respiratory illness rates would be more elevated in children exposed to pollution for several years than in newly arrived residents, and (3) that the accuracy of illness reporting by parents would be similar for all communities.

For each morbidity condition, two analyses were done. In the first, the dependent variable was the percentage of children reporting one or more episodes. For the second, the dependent variable was the percentage of children reporting two or more episodes.

Hypotheses were tested by a general linear model for categorical data.[20] This technique utilizes weighted regression on categorical data and allows estimation of each effect in the model adjusted for all other effects in the model. Significance was ascertained by Chi Square procedures. Covariates used in the model were age (three categories: 1 to 4, 5 to 8, 9 to 12), sex, and educational attainment (two categories: < completed high school, ≥ completed high school), which was an index of socioeconomic status (SES).

## RESULTS

### Exposure to Air Pollutants

The estimated annual pollutant levels for each community for the study period and the decade preceding showed a distinct gradient for sulfur dioxide exposure (Table 3.3.1). Estimated concentrations in the Low I and Low II communities (10 to 26 $\mu g/m^3$) were well within the relevant national ambient air quality standards. Estimated total suspended particulate levels (45 to 55 $\mu g/m^3$) and suspended sulfates (2.9 to 3.3 $\mu g/m^3$) in these two communities were also low. Based on the estimated annual pollutant levels, it appears that air quality in these two communities had varied little during the lifetime of the study population.

The Low III community differed from the other Low exposure communities in that estimated annual average suspended particulates (99 to 115 $\mu g/m^3$) were elevated above the National Primary Air Quality Standard, while estimated suspended sulfates, for which there is no air quality standard, were also somewhat elevated (6.2 to 7.3 $\mu g/m^3$). Moreover, because of industrial emissions, the Low III com-

**Table 3.3.1. ESTIMATED ANNUAL SULFUR DIOXIDE, TOTAL SUSPENDED PARTICULATE, AND SUSPENDED SULFATE LEVELS IN FIVE ROCKY MOUNTAIN COMMUNITIES**

| | Annual pollutant concentrations, $\mu g/m^3$ | | | | | |
|---|---|---|---|---|---|---|
| | Sulfur dioxide | | Total suspended particulate | | Suspended sulfate | |
| Community | During study (1968-70) | Decade prior to study (1958-67)[a] | During study (1968-70) | Decade prior to study (1958-67)[a] | During study (1968-70) | Decade prior to study (1958-67)[a] |
| Low I | 10 | 10 | 50 | 50 | 3.3 | 3.3 |
| Low II | 26 | 26 | 45 | 55 | 4.9 | 4.9 |
| Low III | 67 | 37 | 115 | 99 | 7.3 | 6.2 |
| High I | 177 | 187 | 65 | 63 | 7.2 | 7.0 |
| High II | 374 | 270 | 102 | 146 | 11.3 | 16.2 |

[a] Based on production figures and emissions estimates.

munity probably experienced peak short-term exposures of sulfur dioxide that were considerably higher and more frequent than the two other Low exposure communities. The somewhat higher estimated annual average sulfur levels in the Low III community (37 to 67 $\mu g/m^3$) probably reflected these short-term peak exposures. Ideally, the Low III community would be approached separately in an attempt to isolate the effects of short-term peak exposures to sulfur dioxide and moderately elevated suspended particulates. Unfortunately, the small size of the Low III community prevented such an analysis. On the basis of estimated pollutant exposures, it seems that pooling the Low III community with the two cleaner Low exposure communities is a conservative procedure that would tend to diminish any differences attributable to air pollutants.

It is estimated that the two High exposure communities were both exposed to annual average sulfur dioxide levels (177 to 374 $\mu g/m^3$) well above the relevant air quality standards. The estimated annual average sulfur dioxide levels in the High I community were consistently elevated (177 to 187 $\mu g/m^3$) during the entire lifetimes of the children surveyed. Estimated sulfur dioxide levels in the High II community were higher (270 $\mu g/m^3$) during the infancy and early childhood of the study population, and appeared to increase even more (374 $\mu g/m^3$) during the study. On the other hand, estimated annual average suspended sulfate levels (11.3 to 16.2 $\mu g/m^3$) and total suspended particulate levels (102 to 146 $\mu g/m^3$) in the High II community were distinctly higher than levels in the High I community where total suspended particulates (63 to 65 $\mu g/m^3$) were well within the National Ambient Air Quality Standard and suspended sulfates were less drastically elevated (7.0 to 7.2 $\mu g/m^3$). In none of the communities would one expect to find significant elevations of nitrogen dioxide, suspended nitrates, carbon monoxide, or gaseous hydrocarbons.

## Community Characteristics

Intercommunity differences in reported illness might be influenced by large intercommunity differences in questionnaire completion rates, socioeconomic status, crowding, indoor pollution by cigarette smoking, and exclusions of respondents prior to analysis (Table 3.3.2).

Families from the pooled High and pooled Low exposure communities completed and returned the study questionnaires equally well, and no large

Table 3.3.2. COMMUNITY CHARACTERISTICS: QUESTIONNAIRE COMPLETION RATES, EDUCATIONAL ATTAINMENT OF HEAD OF HOUSEHOLD, CROWDING, AND SMOKING HABITS

| Community | Percent of families completing questionnaire | Percent of household heads who completed high school | Percent of households with ≤1 person per room | Percent of parents who are current cigarette smokers | |
|---|---|---|---|---|---|
| | | | | Mothers | Fathers |
| Pooled Low | 84.9 | 85 | 81 | 33 | 37 |
| Low I | 85.0 | 91 | 87 | 26 | 29 |
| Low II | 84.7 | 80 | 77 | 39 | 48 |
| Low III | 84.9 | 74 | 71 | 41 | 46 |
| Pooled High | 83.5 | 61 | 70 | 42 | 54 |
| High I | 83.7 | 63 | 68 | 44 | 52 |
| High II | 83.3 | 58 | 72 | 40 | 56 |

community differences were noted within the two pollution strata. Significant differences were noted, however, in socioeconomic status as indexed by educational attainment of the head of household. The two High pollution communities were definitely less well educated than the Low exposure communities ($p < 0.001$). Household crowding, an index of family size and economic level, was also significantly more frequent in the High exposure communities ($p < 0.001$). Both mothers and fathers from the High exposure communities were also more likely to be cigarette smokers than mothers and fathers from the Low exposure communities ($p < 0.001$). Fortunately, there were no intercommunity differences in the intensity of cigarette smoking among either mothers ($p > 0.20$) or fathers ($p > 0.10$) who had the habit.

What might be the effect of the differences between the characteristics of High exposure and Low exposure communities? Other studies show that higher socioeconomic status is linked with increased frequency of croup and bronchitis coupled with a decreased frequency of pneumonia. These studies inconsistently associated heavy cigarette smoking by parents with increased bronchitis in their children. Paradoxically, children of light smokers were found to have less croup than children of nonsmokers or heavy smokers who reported similar rates for croup. Clearly, social status and cigarette smoking might bias the present study in a complex fashion. Since cigarette smoking and lower educational attainment are inextricably linked, inclusion of both these variables in an adjustment procedure might cause over-adjustment of illness rates. Furthermore, the sample size and the statistical analysis limited the possible number of adjustments, and a number of other disease determinants, including age, sex, and residential mobility, clearly required it. Preliminary analysis indicated that consistent overall illness differences within pollution strata were attributable to socioeconomic differences. Thus, socioeconomic adjustment was clearly necessary. On the other hand, intercommunity differences in smoking habits were less striking, and the expected effects of smoking on the clinical syndromes that comprise the lower respiratory illness grouping were to some extent counterbalancing. Croup might tend to be modestly increased in the Low exposure communities because of the modest excess of current nonsmokers there. On the other hand, the same factor would tend to slightly accentuate bronchitis rates in the High exposure communities. Therefore, no adjustments were made for differences that might be attributable to cigarette smoking.

As specified in the study protocol, children of the families who completed the health questionnaire were excluded from the study because of missing questionnaire data or presence of active asthma requiring treatment by physician during the study period.

One-tenth of the respondents were excluded because of incomplete or uninterpretable questionnaire responses, and there were no remarkable differences between the High and the Low exposure communities (Table 3.3.3). Almost half of the exclusions were caused by missing data on age and by exclusion of all children under 1 year of age.

## Acute Lower Respiratory Disease Evaluation

### *Asthmatics*

Approximately 5 percent of the respondent population were asthmatics, and these were excluded from the principal analysis. The number of asthmatics proved too small to allow adequate adjustments for other strong determinants of acute lower respiratory disease (Table 3.3.4). Nevertheless, parents living in the High exposure communities consistently reported significantly more total acute lower respiratory illness, croup, and bronchitis among their asthmatic children than did parents from the Low exposure communities. Clinically, the greatest proportionate morbidity excess was among children having the croup syndrome. The frequency of croup and that of hospitalizations exactly paralleled the long-term exposure gradients for sulfur dioxide and suspended particulate sulfates.

Increased croup and hospitalization rates were apparent in the Low III exposure community, but with the small sample size, this excess was not statistically significant. Since asthmatics from both High exposure communities and the Low III community also more frequently reported hospitalizations, they were combined for testing, and excess from these three communities approached statistical significance. The described excesses for croup and hospitalizations could be attributed to repeated short-term peak sulfur dioxide exposures, to peak suspended sulfate exposures, or to longer, lower level exposures of these pollutants. It seems reasonable that exclusion of asthmatics from the principal analysis would lead to a conservative estimate of intercommunity differences attributable to ambient air pollution.

### *Total Acute Respiratory Illness*

After all exclusions, the study population of 5773 was distributed by length of residence so that one could distinguish the exposure duration necessary for the emergence of any respiratory morbidity accompanying high levels of ambient air pollutants (Table 3.3.5). This procedure also isolated any increases in illnesses that may have been associated

**Table 3.3.3. COMMUNITY CHARACTERISTICS: REASONS FOR EXCLUSION OF NONASTHMATIC CHILDREN**

| | Percent of nonasthmatic children excluded | | |
|---|---|---|---|
| Community | Because of missing data on education, sex, or illness | Because of missing data on age | Total |
| Pooled Low | 5 | 6 | 11 |
| Low I | 4 | 8 | 12 |
| Low II | 6 | 4 | 10 |
| Low III | 4 | 5 | 9 |
| Pooled High | 4 | 5 | 9 |
| High I | 3 | 4 | 7 |
| High II | 6 | 6 | 12 |
| Total | 5 | 5 | 10 |

## Table 3.3.4. PERCENT OF ASTHMATIC CHILDREN REPORTING AT LEAST ONE LOWER RESPIRATORY ILLNESS DISTRIBUTED BY COMMUNITY AND CLINICAL DIAGNOSIS

| Community | Number of children | Percent reporting lower respiratory illness[a] | | | | |
|---|---|---|---|---|---|---|
| | | Any lower respiratory disease | Croup | Bronchitis | Pneumonia | Hospitalization |
| Pooled Low | 149 | 52 | 14 | 44 | 17 | 19 |
| Low I | 75 | 52 | 11 | 48 | 12 | 15 |
| Low II | 43 | 51 | 14 | 47 | 23 | 21 |
| Low III | 31 | 52 | 23 | 42 | 23 | 26 |
| Pooled High | 141 | 65 | 23 | 60 | 22 | 25 |
| High I | 54 | 65 | 26 | 61 | 24 | 28 |
| High II | 87 | 64 | 21 | 59 | 21 | 23 |

[a] The cluster of High exposure communities reported significantly more total lower respiratory illness ($p < 0.05$), croup ($p < 0.05$), and bronchitis ($p < 0.01$). If the Low III community were included in the High exposure cluster, hospitalization increases in that cluster would be nearly significant ($0.10 < p < 0.05$).

## Table 3.3.5. NUMBER OF NONASTHMATIC CHILDREN USED IN ALL ILLNESS ANALYSES DISTRIBUTED BY COMMUNITY AND RESIDENCE DURATION

| Community | Number of children | | | |
|---|---|---|---|---|
| | $\leq$1 yr residence | > 1 to < 3 yr residence | $\geq$3 yr residence | Total |
| Pooled Low | 512 | 213 | 1772 | 2497 |
| Low I | 290 | 145 | 786 | 1221 |
| Low II | 148 | 42 | 640 | 830 |
| Low III | 74 | 26 | 346 | 446 |
| Pooled High | 492 | 251 | 2533 | 3276 |
| High I | 180 | 68 | 1505 | 1753 |
| High II | 312 | 183 | 1028 | 1523 |
| Total | 1004 | 464 | 4305 | 5773 |

with the fact of family migration since increases in many types of illness are reported after major changes in life situations.[11] An overwhelmingly important determinant of acute respiratory illness is age, with the highest rates in preschool children (Figure 3.3.1). If the experience of children less than 1 year of age were included, the origin point in Figure 3.3.1 would fall at a point substantially lower than that plotted for 1-year olds. Infants in the first year are in part protected by passive transfer of maternal antibodies and relative isolation within their homes. However, the tremendous excess in lower respiratory illness observed in older preschool children probably did not result from the fading of passive immunity but rather from the fact that the youngest children were not yet born, as were their older siblings, when pandemic influenza swept across the United States in late 1968 and early 1969. Higher attack rates were generally found among boys, but females inched into the lead position at age 12. This observed sex-age pattern is classically reported for many infectious diseases. The important fact is that any attempt to assess pollution effects must adjust for differences in age. Sex adjustment, while desirable, is not as necessary because there was no evidence of intercommunity differences in the sex ratio.

Both unadjusted illness attack rates and rates adjusted for the effects of age, sex, and socioeconomic status were computed for the acute respiratory illness grouping as a whole and for each of its constituent clinical syndromes. Unadjusted data are given in the Appendix, Tables 3.3.A.1 to 3.3.A.5. For adjusted rates, sample size restrictions required frequent pooling. Therefore, residence durations of less than 3 years were combined, as were those of 3 years or more. Three other points deserve emphasis. First, unadjusted illness rates for nonasthmatic children differed from illness rates in asthmatics in that no intercommunity differences were immediately apparent. The second observation was that length of residence was a powerful determinant of illness that fully justified separate consideration in the analysis. The third puzzling point was that the illness behavior of children from families who had recently migrated seemed paradoxical, with children from families moving into the cluster of less polluted communities having more illness during the first years of residence than similar children moving into the more polluted cluster. Thereafter, children from the less polluted communities generally reported progressively lower attack rates, while those from the more polluted cluster reported progressively higher attack rates. It seems possible that families with vulnerable children somehow preferentially avoided moving into the more polluted cluster. Since the presence of chronic respiratory disease symptoms in adults accurately predicted the occurrence of excess lower respiratory disease in their children,[21] perhaps parents of the more susceptible children were prevented from being employed in and then moving into the more polluted cluster by occupational health preplacement screening. Another possible explanation is that the communities in the High pollution cluster are smaller and hence have fewer endogenous strains of infectious organisms that typically affect migrants than the larger, more heterogeneous communities in the Low pollution cluster.

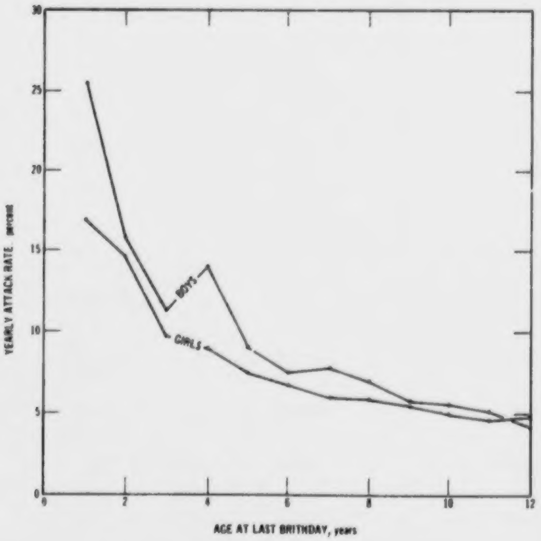

Figure 3.3.1. Age-specific annual mean attack rates for acute lower respiratory illness.

After the rates were appropriately adjusted, excesses in single and repeated attacks of any acute lower respiratory illness were apparent among children living in the High exposure communities for 3 or more years (Table 3.3.6). The significance of each of the major determinants of acute respiratory disease was tested and the results are summarized (Table 3.3.7). Age, sex, and pollution exposure all had significant impacts on attack rates for repeated episodes of lower respiratory illness among children living in the High pollution cluster for 3 or more years. The observed two failures of model fit, which were not themselves independent of one another, could be explained by chance alone if one considered the total number of model fits tested.

**Table 3.3.6. AGE-, SEX-, SES-ADJUSTED 3-YEAR ATTACK RATES FOR ANY LOWER RESPIRATORY ILLNESS BY RESIDENCE DURATION AND NUMBER OF EPISODES**

| Pooled communities[a] | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | <3 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 25.0 | 21.3 | 22.9 |
| High | | 22.0 | 24.2 | 23.8 |
| Low | ≥2 | 9.2 | 9.2 | 9.6 |
| High | | 9.1 | 12.1 | 12.4 |

[a] Low—Low I, Low II, and Low III; High—High I and High II.

**Table 3.3.7. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, ANY LOWER RESPIRATORY DISEASE**

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | < 3 yr | ≥ 3 yr | Any length |
| Exposure | ≥ 1 | NS | <0.10 | NS |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | <0.001 | <0.001 |
| Socioeconomic status | | NS | NS | <0.05 |
| Fit of model | | NS | NS | NS |
| Exposure | ≥ 2 | NS | <0.001 | <0.01 |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | <0.001 | <0.001 |
| Socioeconomic status | | <0.01 | NS | <0.01 |
| Fit of model | | <0.02 | NS | <0.05 |

[a] NS—not significant, $p > 0.10$.

### *Croup*

Excess laryngotracheobronchitis, or croup, morbidity in the High pollution cluster accounted for a large portion of the previously described excess in total acute lower respiratory illness (Table 3.3.8). Pollution effects were significant in explaining higher attack rates for single or repeated episodes of illness (Table 3.3.9). New arrivals in the more polluted communities reported less illness than those in the cleaner communities. Younger children, especially those under 4, and girls had higher attack rates. Socially advantaged families also reported more croup than less advantaged families.

### *Bronchitis*

Appropriately adjusted bronchitis rates trended in the same direction as those for total lower respiratory illness (Table 3.3.10). However, for bronchitis the differences were not significant (Table 3.3.11). The

Table 3.3.8. AGE-, SEX-, SES-ADJUSTED 3-YEAR ATTACK RATES FOR CROUP BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Pooled communities[a] | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | <3 yr residence | ≥ 3 yr residence | Any length residence |
| Low | ≥1 | 8.1 | 6.2 | 7.2 |
| High | | 7.7 | 11.2 | 10.5 |
| Low | ≥2 | 2.4 | 3.1 | 3.2 |
| High | | 2.2 | 4.8 | 4.3 |

[a] Low—Low I, Low II, Low III; High—High I and High II.

Table 3.3.9. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, CROUP

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | < 3 yr | ≥3 yr | Any length |
| Exposure | ≥1 | NS | < 0.001 | < 0.001 |
| Age | | <0.001 | < 0.001 | < 0.001 |
| Sex | | <0.02 | < 0.01 | < 0.01 |
| Socioeconomic status | | NS | NS | < 0.02 |
| Fit of model | | NS | NS | NS |
| Exposure | ≥2 | NS | < 0.05 | <0.10 |
| Age | | < 0.05 | < 0.001 | < 0.001 |
| Sex | | NS | < 0.01 | < 0.02 |
| Socioeconomic status | | <0.10 | NS | < 0.10 |
| Fit of model | | NS | NS | NS |

[a] NS—not significant, $p > 0.10$.

Table 3.3.10. AGE-, SEX-, SES-ADJUSTED 3-YEAR ATTACK RATES FOR BRONCHITIS BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Pooled communities[a] | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | < 3 yr residence | ≥3 yr residence | Any length residence |
| Low | ≥1 | 16.2 | 14.9 | 15.6 |
| High | | 14.5 | 15.8 | 15.5 |
| Low | ≥2 | 5.3 | 5.1 | 5.2 |
| High | | 4.7 | 6.7 | 6.3 |

[a] Low—Low I, Low II, and Low III; High—High I and High II.

**Table 3.3.11. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, BRONCHITIS**

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | $< 3$ yr | $\geq 3$ yr | Any length |
| Exposure | $\geq 1$ | NS | NS | NS |
| Age | | $< 0.001$ | $< 0.001$ | $< 0.001$ |
| Sex | | NS | $< 0.001$ | $< 0.01$ |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |
| Exposure | $\geq 2$ | NS | $< 0.10$ | NS |
| Age | | $< 0.01$ | $< 0.01$ | $< 0.001$ |
| Sex | | NS | $< 0.01$ | $< 0.001$ |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |

[a] NS—not significant, $p > 0.10$.

direction of the differences was hardly surprising since acute bronchitis is the largest single component of the total lower respiratory illness grouping. Again, children in the cleaner communities tend to have more illness initially but less after 3 years than children from polluted communities. Significant differences attributable to age and sex again occurred. As with croup, more advantaged families tended, although not significantly, to report higher attack rates.

### *Pneumonia*

Attack rates for single episodes of pneumonia mimicked the community pollution, age, and sex trends previously described (Table 3.3.12). Excesses in the more polluted communities, though not statistically significant (Table 3.3.13), are nevertheless worrisome since pneumonia in children is a serious, potentially life-threatening illness. Fortunately, practically no excess in repeated episodes of pneumonia was observed. Socioeconomic effects, while not significant, did trend opposite to those observed for croup and bronchitis in that children from less advantaged families had higher pneumonia attack rates.

### *Hospitalizations*

The nonsignificant trend toward more frequent hospitalizations in the polluted communities, previously found among asthmatics, was replicated in the larger nonasthmatic population (Tables 3.3.14 and 3.3.15). Moreover, a nonsignificant tendency toward higher rates for repeated hospitalization was apparent in the more polluted communities, and this finding gives warning that the excess in illness induced in the more polluted communities may be more severe. The effects of age, sex, and socioeconomic status paralleled those described for penumonia, as might be expected since many childhood pneumonia cases frequently require hospital care.

### *Age-specific Attack Rates*

Because croup appeared to be the clinical syndrome most likely induced, at least in part, by sulfur dioxide and suspended sulfate exposures, an attempt was made to link exposure duration with the onset of higher attack rates. It was necessary to attain adequate sample sizes and to isolate, balance, or adjust for the duration of residence, for age, and for

### Table 3.3.12. AGE-, SEX-, SES-ADJUSTED 3-YEAR ATTACK RATES FOR PNEUMONIA BY RESIDENCE DURATION AND NUMBER OF EPISODES

| Pooled communities[a] | Number of illness episodes | Attack rate, percent | | |
|---|---|---|---|---|
| | | $<3$ yr residence | $\geq 3$ yr residence | Any length residence |
| Low | $\geq 1$ | 5.0 | 5.2 | 5.4 |
| High | | 6.7 | 5.7 | 6.0 |
| Low | $\geq 2$ | 1.1 | 1.4 | 1.2 |
| High | | 1.4 | 1.4 | 1.3 |

[a] Low—Low I, Low II, and Low III; High—High I and High II.

### Table 3.3.13. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, PNEUMONIA

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] | | |
|---|---|---|---|---|
| | | $<3$ yr | $\geq 3$ yr | Any length |
| Exposure | $\geq 1$ | NS | NS | NS |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | <0.01 | <0.05 |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |
| Exposure | $\geq 2$ | NS | NS | NS |
| Age | | <0.01 | <0.01 | <0.01 |
| Sex | | NS | <0.02 | <0.02 |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |

[a] NS—not significant, $p > 0.10$.

sex. The problem of preventing socioeconomic bias further complicated the issue. A simple analytical scheme was devised, and the influence of intervening variables was assessed prior to testing for significance. The analysis was limited to children of families who had lived for 3 or more years in the same study community. The population was partitioned according to community air pollution, the sexes were combined, and adjacent ages were pooled. No sex or age biases were apparent in the two resulting distributions. Any bias attributable to overreporting among the more advantaged families would hamper efforts to attribute intercluster differences to ambient air pollution since the Low pollution cluster contained proportionately more of the advantaged families. Age-specific attack rates for each cluster were then plotted (Figure 3.3.2). Community clusters had practically identical attack rates at ages 1 and 2, but never thereafter. Attack rates for children from the more polluted cluster decreased much more slowly than those for children from the less polluted community cluster. The gap between the two clusters

### Table 3.3.14. AGE-, SEX-, SES-ADJUSTED 3-YEAR HOSPITALIZATION RATES BY RESIDENCE DURATION AND NUMBER OF ADMISSIONS

| Pooled communities[a] | Number of illness episodes | Residence duration <br> < 3 yr residence | ≥ 3 yr residence | Any length residence |
|---|---|---|---|---|
| Low | ≥ 1 | 4.3 | 4.3 | 4.8 |
| High | | 6.3 | 5.4 | 5.7 |
| Low | ≥ 2 | 1.2 | 1.3 | 1.0 |
| High | | 1.6 | 1.4 | 1.2 |

[a] Low—Low I, Low II, and Low III; High—High I and High II.

### Table 3.3.15. ANALYSIS OF VARIANCE FOR CATEGORICAL DATA: SUMMARY OF PROBABILITIES BY NUMBER OF EPISODES AND RESIDENCE DURATION, HOSPITALIZATION

| Factor | Number of episodes | Probability of effect for indicated residence duration[a] <br> < 3 yr | ≥ 3 yr | Any length |
|---|---|---|---|---|
| Exposure | ≥1 | NS | NS | NS |
| Age | | <0.001 | <0.001 | <0.001 |
| Sex | | NS | <0.01 | <0.05 |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |
| Exposure | ≥2 | NS | NS | NS |
| Age | | NS | <0.001 | <0.001 |
| Sex | | NS | <0.02 | <0.01 |
| Socioeconomic status | | NS | NS | NS |
| Fit of model | | NS | NS | NS |

[a] NS—not significant, $p > 0.10$.

appeared to be closing by age 11 to 12. Statistically significant increases were demonstrable in the High pollution areas by age 3 to 4. There can be little doubt, then, that an exposure duration of 3 years was strongly coupled to increased croup morbidity.

Attack rates within 4-year age groupings (1 to 4, 5 to 8, and 9 to 12) for families having different educational levels were computed to look for any peculiar interactions involving age, social status, and the two most common lower respiratory illness syndromes: croup and bronchitis. A relative risk related to pollution exposure was then constructed using the appropriate attack rate for the more polluted communities as the numerator and the attack rate for the cleaner communities as the denominator (Figure 3.3.3). The full impact of intercommunity differences in social class became clear. Pollution had a greater relative effect on less advantaged families than on advantaged families. This excess was most marked when croup was considered. One factor contributing to such an excess may well be the poorer housing of less advantaged families. Such housing would more likely be located closer to

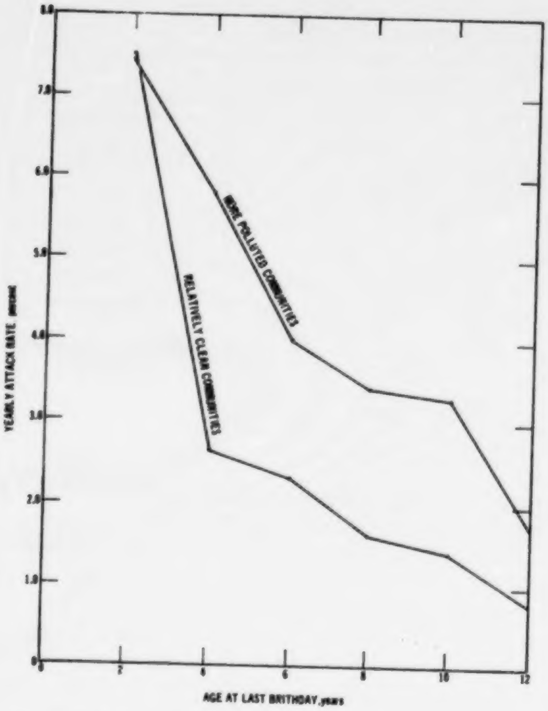

Figure 3.3.2. Age-specific annual mean attack rates for croup distributed by community air pollution.

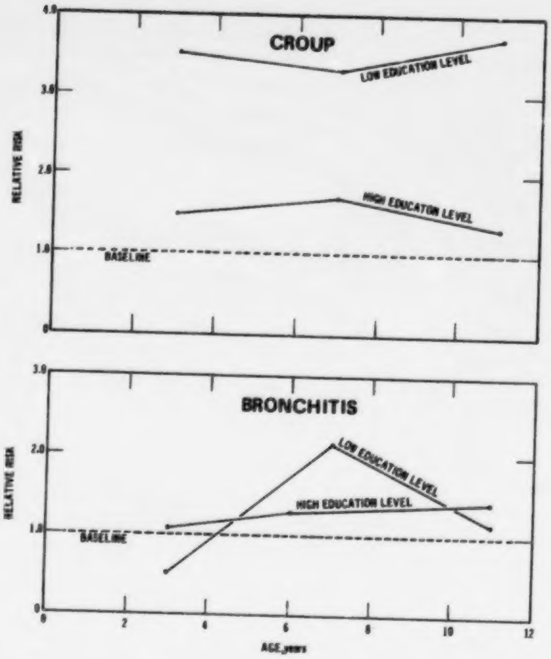

Figure 3.3.3. Difference in the relative risk of croup and bronchitis which can be attributed to residence in polluted communities.

the point sources of pollution and might well be less well constructed, allowing freer penetration by pollutants. Poorer families are also less likely to have modern heating and air conditioning systems, which may in part dampen the penetration of air pollutants into the home.

### *Physician Diagnostic Patterns and Validating Questionnaire-reported Illness Using Physician Records*

Possible effects of differences in diagnostic habits of practicing physicians or overreporting of acute lower respiratory illness on the significant morbidity excesses thus far attributed to ambient air pollutants were considered. There is no evidence, however, for the occurrence of such a bias, and much evidence against it.

Eighteen physicians who cared for children who comprised the study population were asked to diagnose six respiratory syndromes as "bronchitis" or "not bronchitis." The syndromes were graded ranging from pharyngitis with minimal, but unmistakable, lower respiratory symptoms to classical pneumonia. Bronchitis was purposely chosen since bronchitis in children is a somewhat imprecise diagnosis, thus allowing a better chance to discover any latent intercommunity differences. Physicians' diagnostic decisions were tallied for pooled communities (Table 3.3.16). No remarkable intercommunity differences could be attributed to differences in diagnostic custom. Physicians, as expected, agreed with each other better at the extremes of the syndrome and tended to disagree when the clinical picture was less distinctive.

The questionnaire used in this study classified deep chest colds as bronchitis, which is a lower respiratory illness. This made validation of questionnaire responses a difficult task since physicians will often note only acute respiratory infection or upper respiratory illness when, in fact, they diagnose a "chest cold." At times, physicians only record symptoms which are of value in helping recall the

**Table 3.3.16. CLINICAL DEFINITION OF ACUTE BRONCHITIS: NUMBER OF PHYSICIANS CHARACTERIZING SIX RESPIRATORY ILLNESS SYNDROMES AS "BRONCHITIS" OR "NOT BRONCHITIS"**

| Syndrome description | Physicians from Low exposure areas | | Physicians from High exposure areas | |
|---|---|---|---|---|
| | Bronchitis | Not bronchitis | Bronchitis | Not bronchitis |
| Pharyngitis | 0 | 3 | 3 | 12 |
| Deep chest cold or mild bronchitis | 3 | 0 | 13 | 2 |
| Croup or laryngotracheobronchitis | 2 | 1 | 4 | 11 |
| Classical bronchitis | 3 | 0 | 11 | 4 |
| Severe bronchitis or early pneumonia | 1 | 2 | 6 | 9 |
| Pneumonia | 0 | 3 | 0 | 15 |

patient's medical history. Other possible errors include imperfections in memory, diagnosis, doctor-patient communication, and enumeration. For the validation estimate, either the specific diagnosis of bronchitis or any acute respiratory illness confirmed by a medical record was accepted as validation of lower respiratory illness reported by the questionnaire respondents. On the other hand, a questionnaire reporting no illness was not considered in error unless a specific lower respiratory illness syndrome was diagnosed. For each area, a 15 percent sample of questionnaire responses was validated by the described review of medical records. For each physician included in the validation sample, children sick with bronchitis and an appropriate number of children denying lower respiratory illness were selected. (For this reason, the use of sensitivity and specificity measures to arrive at corrected rates is not valid.)[22] Within the "sick sample" attempts were made to validate all lower respiratory illness episodes. There was a trend toward poorer validation of questionnaire-reported illness in the High pollution communities (Table 3.3.17). However, within the "well sample," there was closer agreement with physician records in the High pollution communities. While probably not of sufficient magnitude to bias the study, these trends were disturbing and difficult to interpret.

## DISCUSSION

For nonasthmatic children, elevated annual average exposures, and perhaps repeated short-term peak exposures, to sulfur dioxide and suspended sulfates were significantly associated with excesses in repeated episodes of any lower respiratory disease. The same significant association was found for single and repeated episodes of croup. Nonsignificant morbidity excesses in bronchitis, pneumonia, and hospitalizations because of acute lower respiratory illness were also found in the High exposure cluster (Table 3.3.18). The analysis also revealed that suspected determinants of acute respiratory disease, including age, sex, social class, and change in family situation, were behaving in predictable fashion.[1,2,23-26] Once this was assured, construction of an appropriate age-specific model for croup, which was the disorder most influenced by pollution, was easily accomplished. The model clearly established that 3- to 4-year exposures to elevated annual average concentrations of sulfur dioxide (177 to 374 $\mu g/m^3$) accompanied by elevated annual average levels of suspended sulfates (7 to 11 $\mu g/m^3$) would enhance host susceptibility for croup. It cannot be categorically stated that this effect was not attributable to repeated, short-term peak exposures that were much higher, though this seems unlikely because of the

### Table 3.3.17. VALIDATION OF QUESTIONNAIRE RESPONSES REPORTING CHILDREN EITHER SICK OR WELL AGAINST PHYSICIAN RECORDS[a]

| | Any lower respiratory illness | | | | Bronchitis | | | |
|---|---|---|---|---|---|---|---|---|
| | Sick validated | | Well validated | | Sick validated | | Well validated | |
| Community | Percent | n[b] | Percent | n | Percent | n | Percent | n |
| Pooled Low | 88 | 86 | 75 | 191 | 80 | 77 | 76 | 200 |
| Low I | 90 | 42 | 78 | 77 | 84 | 37 | 77 | 82 |
| Low II | 92 | 26 | 74 | 68 | 87 | 23 | 78 | 71 |
| Low III | 78 | 18 | 74 | 46 | 71 | 17 | 81 | 47 |
| Pooled High | 75 | 108 | 85 | 162 | 71 | 91 | 93 | 180 |
| High I | 70 | 61 | 84 | 85 | 65 | 51 | 94 | 96 |
| High II | 83 | 47 | 86 | 77 | 78 | 40 | 92 | 84 |
| Overall | 81 | 194 | 80 | 353 | 76 | 168 | 85 | 380 |

[a] Allowances were made to adjust for multiplicity of diagnostic terms used to describe mixed upper and lower respiratory infections. Otherwise, the agreements would be substantially reduced.

[b] n is the number of records examined.

relatively long latent period before the effect became manifest. Quantifying the adverse impact of pollutants in children who were not asthmatics was much more difficult than in asthmatic children, as the statistical analysis was forced to cope with bothersome intercommunity differences in social class as well as with migration, which was probably itself influenced by pollution. The investigators had carefully attempted to avoid any intercommunity social class differences, but were constrained by the paucity of suitable geographically and climatologically similar communities. Moreover, grouping of the Low III smelter community with the Low pollution cluster, while perfectly justified by its relatively low annual average sulfur dioxide level, was a course selected from a narrow array of poor options. We believe this led to a conservative estimate of pollution effects. Appropriate adjustments were able to cope with the confusing effects of intervening variables, and a consistent morbidity pattern became apparent.

For asthmatic children, elevated sulfur dioxide and suspended sulfate air pollution was significantly linked to excess morbidity for the acute respiratory grouping as a whole and for croup and bronchitis separately. As with nonasthmatic children, nonsignificant morbidity excesses in pneumonia and hospitalizations because of acute lower respiratory illness were found in the High exposure cluster (Table 3.3.18).

What limitations still might alter the conclusions of the present study? Overreporting and nonrespondent bias are two. The worst projectable overreporting possible, ignoring compensating socioeconomic effects, might reduce the magnitude of the observed excesses but could not begin to equalize the large excess morbidity observed.

It is also quite unlikely that the study results would have changed if the children with missing information (10 percent) and the nonrespondents (15 percent) had been included in the analysis. One can calculate how the inclusion of these children might have affected the present study outcome. To equal the significantly higher rates for any lower respiratory disease in the High communities, a greater than two-fold increase in the rate among Low community nonrespondents coupled with a rate of zero among High community nonrespondents would have been necessary. Alternately, a 50 percent increase among the Low community nonrespondents along with a similar decrease for those in the High community

**Table 3.3.18. EXCESS ACUTE LOWER RESPIRATORY ILLNESS AMONG CHILDREN EXPOSED 3 OR MORE YEARS TO ELEVATED LEVELS OF SULFUR DIOXIDE**

| Illness | Number of illness episodes | Excess in high exposure communities, percent | |
|---|---|---|---|
| | | Among asthmatics | Among nonasthmatics[a] |
| Any acute respiratory disease | ≥1 | 25 | 14 |
| | ≥2 | NA[b] | 42 |
| Croup | ≥1 | 60 | 81 |
| | ≥2 | NA | 55 |
| Bronchitis | ≥1 | 36 | 6 |
| | ≥2 | NA | 31 |
| Pneumonia | ≥1 | 27 | 10 |
| | ≥2 | NA | 0 |
| Hospitalization | ≥1 | 31 | 26 |
| | ≥2 | NA | 8 |

[a] Adjusted for age, sex, and socioeconomic status.

[b] NA—not available.

would have made the overall rates equal. To equal the significantly higher rates for croup, the Low community nonrespondent rate would have to be increased 2.9 times at the same time that the rate for the High community nonrespondents was zero. Rates would also have to be more than 75 percent higher for Low community nonrespondents and more than 75 percent lower among those of the High community for the reported rates of croup to be equal. Such outcomes would be extremely uncommon.

Based on the above analyses and considerations, we conclude that excessive acute lower respiratory illnesses can be expected among asthmatic and nonasthmatic children who are exposed for longer than 2 years to elevated annual average sulfur dioxide levels (177 $\mu g/m^3$) accompanied by elevated annual average suspended sulfate levels (7.2 $\mu g/m^3$) in the presence of low levels of suspended particulates (65 $\mu g/m^3$). We also find strongly suggestive evidence that excess lower respiratory illness in asthmatics can be linked to even lower pollution levels: annual average sulfur dioxide levels as low as 67 $\mu g/m^3$ accompanied by elevations of annual average levels of total suspended particulates (99 to 115 $\mu g/m^3$) and intermediate suspended sulfates (6 to 7 $\mu g/m^3$). Stringent control of sulfur dioxide, suspended particulates, and suspended sulfates should be accompanied by tangible reductions in lower respiratory illness in all children.

## SUMMARY

Acute respiratory morbidity in asthmatic children began to increase with exposures to low annual average levels of sulfur dioxide (67 $\mu g/m^3$) when moderately elevated levels of total suspended particulates (99 to 115 $\mu g/m^3$) were present. Significant excesses in acute lower respiratory illnesses, especially croup, appeared among asthmatic and nonasthmatic children exposed to higher pollution levels for 3 or more years. Such levels involved estimated annual average sulfur dioxide levels of 177 $\mu g/m^3$ accompanied by low estimated annual average levels of particulates (65 $\mu g/m^3$) and minimally elevated estimated average levels of suspended sulfates (7.2 $\mu g/m^3$). There was also suggestive evidence that more serious infections, including pneumonia, that might require hospitalization tended to be more frequent in children exposed to these elevated pollution levels. Other determinants of respiratory illness included age, sex, social status, and recent intercommunity migration. Future studies will be required to validate the morbidity reductions that should follow pollution control.

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### Table 3.3.A.1. UNADJUSTED 3-YEAR ATTACK RATES FOR ANY LOWER RESPIRATORY DISEASE BY NUMBER OF EPISODES AND DURATION OF RESIDENCE

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | >1 to <3 yr residence | ≥3 yr residence | Any length residence |
| Pooled Low | ≥1 | 27.3 | 17.4 | 19.2 | 20.7 |
| Low I | | 27.9 | 17.9 | 20.7 | 22.1 |
| Low II | | 29.7 | 19.0 | 16.7 | 19.2 |
| Low III | | 20.3 | 13.0 | 20.5 | 20.0 |
| Pooled High | ≥1 | 19.3 | 24.7 | 21.3 | 21.2 |
| High I | | 20.6 | 14.7 | 21.7 | 21.3 |
| High II | | 18.6 | 28.4 | 20.7 | 21.2 |
| Pooled Low | ≥2 | 11.5 | 9.9 | 8.5 | 9.2 |
| Low I | | 13.4 | 11.0 | 9.5 | 10.6 |
| Low II | | 11.5 | 7.1 | 7.8 | 8.4 |
| Low III | | 4.1 | 7.7 | 7.2 | 6.7 |
| Pooled High | ≥2 | 9.1 | 9.2 | 11.6 | 11.1 |
| High I | | 8.3 | 5.9 | 12.9 | 12.2 |
| High II | | 9.6 | 10.4 | 9.8 | 9.8 |

### Table 3.3.A.2. UNADJUSTED 3-YEAR ATTACK RATES FOR CROUP BY NUMBER OF EPISODES AND DURATION OF RESIDENCE

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | >1 to <3 yr residence | ≥3 yr residence | Any length residence |
| Pooled Low | ≥1 | 9.2 | 6.1 | 5.4 | 6.2 |
| Low I | | 9.3 | 7.6 | 6.1 | 7.0 |
| Low II | | 10.8 | 2.4 | 4.2 | 5.3 |
| Low III | | 5.4 | 3.8 | 6.1 | 5.8 |
| Pooled High | ≥1 | 5.7 | 10.0 | 9.5 | 9.0 |
| High I | | 3.3 | 7.4 | 10.3 | 9.5 |
| High II | | 7.1 | 10.9 | 8.4 | 8.4 |
| Pooled Low | ≥2 | 3.1 | 1.4 | 2.8 | 2.7 |
| Low I | | 3.8 | 2.1 | 2.9 | 3.0 |
| Low II | | 2.7 | 0.0 | 2.3 | 2.3 |
| Low III | | 1.4 | 0.0 | 3.2 | 2.7 |
| Pooled High | ≥2 | 1.8 | 4.0 | 4.3 | 4.0 |
| High I | | 1.7 | 2.9 | 5.7 | 5.2 |
| High II | | 1.9 | 4.4 | 2.3 | 2.5 |

## Table 3.3.A.3. UNADJUSTED 3-YEAR ATTACK RATES FOR BRONCHITIS BY NUMBER OF EPISODES AND DURATION OF RESIDENCE

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | >1 to <3 yr residence | ≥3 yr residence | Any length residence |
| Pooled Low | ≥1 | 17.4 | 12.2 | 13.9 | 14.5 |
| Low I | | 18.3 | 11.0 | 14.8 | 15.2 |
| Low II | | 16.9 | 16.7 | 12.7 | 13.6 |
| Low III | | 14.9 | 11.5 | 14.2 | 14.1 |
| Pooled High | ≥1 | 14.0 | 14.7 | 14.6 | 14.7 |
| High I | | 15.6 | 7.4 | 14.9 | 14.7 |
| High II | | 13.1 | 17.5 | 14.2 | 14.4 |
| Pooled Low | ≥2 | 6.4 | 5.6 | 4.8 | 6.0 |
| Low I | | 8.6 | 5.5 | 5.2 | 6.1 |
| Low II | | 4.1 | 4.8 | 4.5 | 4.5 |
| Low III | | 2.7 | 7.7 | 4.3 | 4.3 |
| Pooled High | ≥2 | 5.1 | 4.4 | 6.3 | 6.0 |
| High I | | 5.0 | 1.5 | 6.6 | 6.3 |
| High II | | 5.1 | 5.5 | 5.7 | 5.6 |

## Table 3.3.A.4. UNADJUSTED 3-YEAR ATTACK RATES FOR PNEUMONIA BY NUMBER OF EPISODES AND DURATION OF RESIDENCE

| Community | Number of illness episodes | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤1 yr residence | >1 to <3 yr residence | ≥3 yr residence | Any length residence |
| Pooled Low | ≥1 | 6.4 | 4.7 | 4.2 | 4.7 |
| Low I | | 6.6 | 6.2 | 3.9 | 4.8 |
| Low II | | 8.1 | 2.4 | 4.5 | 5.1 |
| Low III | | 2.7 | 0.0 | 4.3 | 3.8 |
| Pooled High | ≥1 | 9.6 | 5.2 | 4.6 | 5.2 |
| High I | | 8.9 | 2.9 | 4.9 | 5.2 |
| High II | | 10.4 | 6.0 | 4.2 | 5.2 |
| Pooled Low | ≥2 | 1.2 | 0.5 | 1.0 | 1.0 |
| Low I | | 1.0 | 0.7 | 1.5 | 1.3 |
| Low II | | 2.0 | 0.0 | 0.6 | 0.8 |
| Low III | | 0.0 | 0.0 | 0.6 | 0.4 |
| Pooled High | ≥2 | 1.4 | 1.6 | 1.1 | 1.2 |
| High I | | 1.7 | 1.5 | 1.1 | 1.2 |
| High II | | 1.3 | 1.6 | 1.0 | 1.1 |

## Table 3.3.A.5. UNADJUSTED 3-YEAR RATES FOR HOSPITALIZATION BY NUMBER OF ADMISSIONS AND DURATION OF RESIDENCE

| Community | Number of illness admissions | Attack rate, percent | | | |
|---|---|---|---|---|---|
| | | ≤ 1 yr residence | >1 to < 3 yr residence | ≥ 3 yr residence | Any length residence |
| Pooled Low | ≥ 1 | 4.5 | 3.3 | 3.6 | 3.8 |
| Low I | | 4.5 | 2.8 | 2.5 | 3.0 |
| Low II | | 4.7 | 7.1 | 4.7 | 4.8 |
| Low III | | 4.1 | 0.0 | 4.0 | 3.8 |
| Pooled High | ≥ 1 | 5.5 | 6.0 | 4.3 | 4.6 |
| High I | | 6.7 | 2.9 | 5.0 | 5.1 |
| High II | | 4.8 | 7.1 | 3.2 | 4.0 |
| Pooled Low | ≥ 2 | 0.6 | 0.0 | 0.8 | 0.7 |
| Low I | | 1.0 | 0.0 | 0.6 | 0.7 |
| Low II | | 0.0 | 0.0 | 1.1 | 0.8 |
| Low III | | 0.0 | 0.0 | 0.9 | 0.7 |
| Pooled High | ≥ 2 | 1.4 | 1.2 | 0.9 | 1.0 |
| High I | | 1.7 | 1.5 | 1.2 | 1.3 |
| High II | | 1.3 | 1.1 | 0.4 | 0.7 |

# CHAPTER 4
# CHICAGO-NORTHWEST INDIANA STUDIES

# 4.1 HUMAN EXPOSURE TO AIR POLLUTANTS IN THE CHICAGO-NORTHWEST INDIANA METROPOLITAN REGION, 1950-1971

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## INTRODUCTION

In order to carry out epidemiologic studies on the health effects of ambient pollution, it is important to have measurements of previous air pollution exposures for the community under study as well as continuing air monitoring surveillance. In the Chicago-Northwest Indiana area, such data were available from the City of Chicago's 20-station air monitoring network and from the Environmental Protection Agency's (EPA) National Air Surveillance Network (NASN).

Two studies, concerned with the effects of sulfur dioxide and total suspended particulates on the health of selected groups, were carried out in the Chicago-Northwest Indiana area during 1969-1970. The Chicago-Northwest Indiana area was chosen as the setting for these studies on the basis of the following criteria: (1) pollution originated from numerous sources, (2) pollutant levels were relatively high for both total suspended particulates (TSP) and sulfur dioxide ($SO_2$), (3) recorded total suspended particulates for 1969 showed an exposure gradient within Metropolitan Chicago, and (4) air monitoring networks were available for both Chicago and outlying areas.

One health effects study was concerned with the acute respiratory disease experience of nursery school children and their families within the City of Chicago.[1] Participating families living within 1.5 miles of one of the city's 20 monitoring stations were grouped into three exposure categories (Highest, High, Intermediate) based on 1969 levels of total suspended particulates. The distribution of the monitoring stations in the City of Chicago is shown in Figure 4.1.1.

In the other study, differences in the prevalence of chronic respiratory disease symptoms were examined in military recruits from the Chicago-Northwest Indiana Metropolitan area.[2] Using reported residential histories and known or estimated pollution levels, subjects were assigned to one of three community exposure strata: relatively clean outlying areas (Low); Chicago suburbs (High II); or the urban core of the Chicago-Northwest Indiana area (High I). The relative location of these areas is shown in Figure 4.1.2.

These two studies were preliminary to the full development of the national Community Health and Environmental Surveillance System as described elsewhere.[3,4] The main objective of the two Chicago studies was to determine health effects of combined exposure to particulates and sulfur oxides. The purpose of this paper is to document the air quality data from which exposure estimates were made.

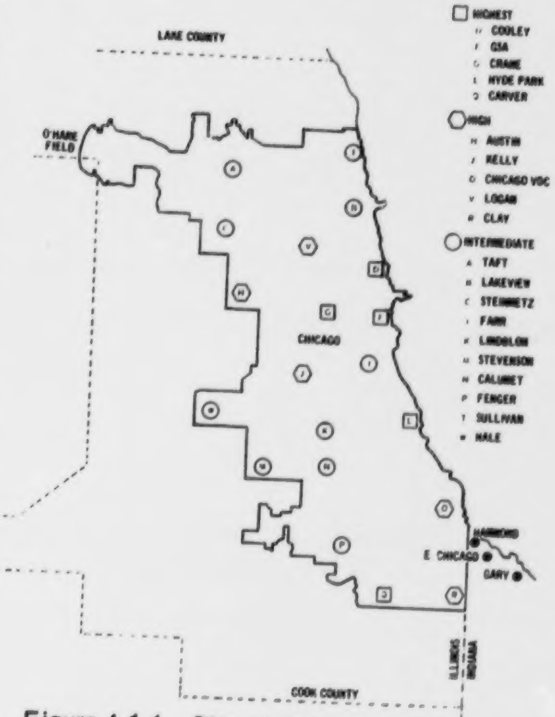

Figure 4.1.1. City of Chicago monitoring stations (City).

## TOPOGRAPHY

Chicago is situated on a flat, or at most gently rolling, plain with an average elevation of 620 feet above mean sea level. While the terrain itself is not rough enough to significantly affect airflow, discontinuities created by local heat sources and sinks do affect low-level mixing. This discontinuity is caused by the large water body mass of Lake Michigan and results in lake breezes in the spring and less strong land breezes in the fall. The central Loop area and most of the lake shore have an additional discontinuity in airflow caused by high-rise apartment buildings. Just how much this affects mixing and dispersion of pollution is not fully known at this time.

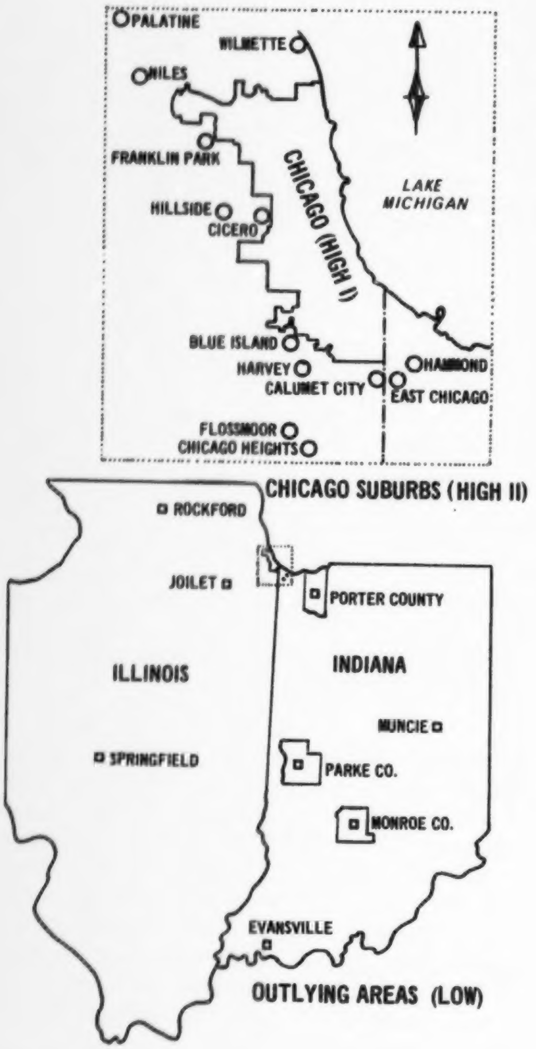

Figure 4.1.2. Chicago-Northwest Indiana Metropolitan area monitoring stations (NASN).

## CLIMATOLOGY

The climate of Chicago is predominately continental, although it is modified to some extent by the proximity of Lake Michigan. The weather, influenced by warm, humid air masses originating from the Gulf of Mexico, by cold, relatively dry air flowing out of the Polar and Arctic regions, and by more moderate masses of air with origins in the North Pacific Ocean, is frequently changeable. Daily temperature ranges are rather large. Annually, the temperature averages 58.7°F. Seasonally, temperatures of 99°F or higher are recorded at Midway Airport in about one-half of the summers and temperatures of -10°F are recorded during one-half of the winters. July is normally the warmest month and January the coldest. The normal heating season is from mid-September to early June, while the air conditioning season is usually from mid-June to early September.

Precipitation averages 33.23 inches a year, with summer the wettest season and winter the driest. Sunshine amounts are generally moderate in summer but quite low in winter due to considerable cloudiness produced locally by lake effects from Lake Michigan. Visibility reductions are frequent because of the availability of adequate water vapor combined with hygroscopic aerosols.

Wind rose summaries for both Midway Airport and O'Hare Field are shown in Figures 4.1.3 and 4.1.4. Normally, the winds generated by the warm season lake breeze reach only a few miles inland. A well developed breeze during the warm season may reduce temperatures near the shore by more than 10°F relative to those further inland.

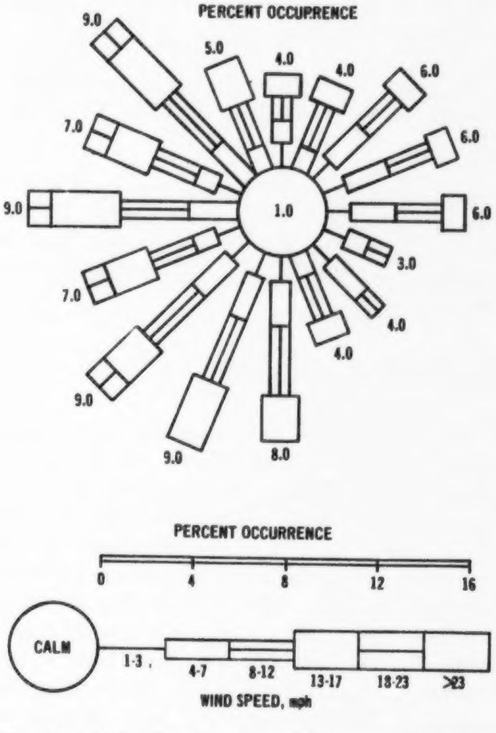

Figure 4.1.3. Surface climatologic wind rose, Chicago Midway Airport, 1951-1960.

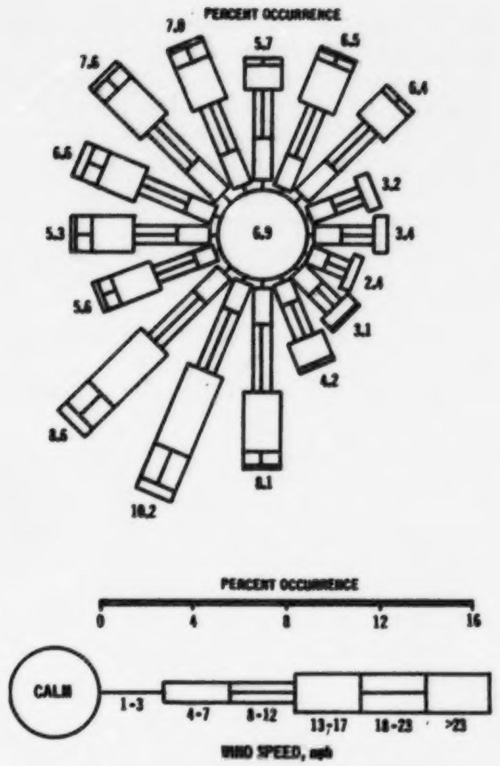

Figure 4.1.4. Surface climatologic wind rose, Chicago O'Hare Airport, 1951-1960.

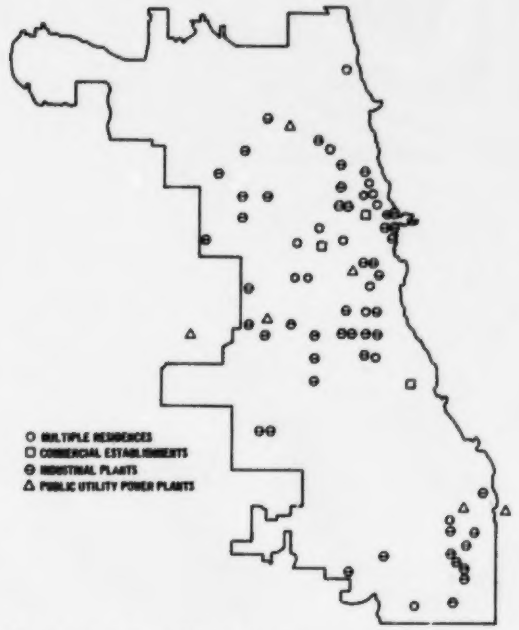

Figure 4.1.5. Major air pollution sources in the City of Chicago.

The climate of suburban and outlying areas of the Chicago-Northwest Indiana Metropolitan area is not greatly affected by Lake Michigan but is generally typical of the Northeastern Great Plains area of the United States.

## POLLUTION SOURCES

Pollution sources in general are most prevalent near the Loop area of Chicago and decrease outwardly away from Lake Michigan. Distribution and type of air pollution sources are shown in Figure 4.1.5. These sources are composed primarily of power generating plants, residential and commercial sources, automobiles, and a variety of industrial sources that are not dominated by any one specific type of product. Pollution effluents from these multiple sources are continually influenced by changing climatological conditions, creating both complex and varying exposures to area residents. Because of the natural attraction for people to live in close proximity to their place of employment, the higher pollution areas are also the most densely populated.

Figure 4.1.6 shows total sulfur dioxide source distribution during 1967. Nonutility industries contributed approximately 10 percent of the total sulfur dioxide emission. This is in sharp contrast to the power generation industry, which contributed 67 percent. Residential areas contributed approximately 15 percent of the total emissions during the same year. Sulfur dioxide emission from residential space heating is given in Figure 4.1.7.

Great progress has been made by the City of Chicago in both reducing emissions and obtaining air quality data throughout the city. Industry and power generating companies have assisted in emissions reduction by both utilizing controls and converting to dual fuel capabilities. The overall effectiveness of these measures is readily apparent when current annual pollutant concentrations are compared with prior concentrations (Figure 4.1.8).

Although significant progress has been made in improving the general air quality in the Chicago area, it should be noted that total suspended particulate levels even in the Intermediate area exceeded the National Primary Air Quality Standard of 75 $\mu g/m^3$ in 1971. Further improvements in air quality will be

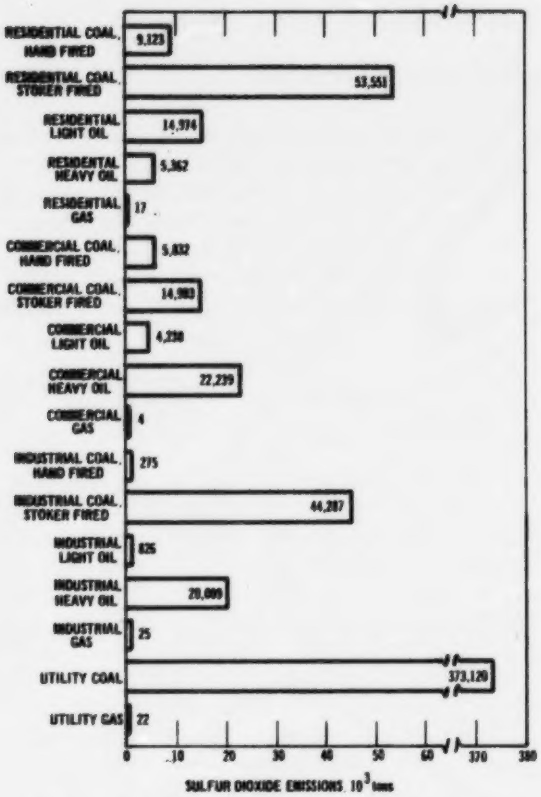

Figure 4.1.6. Annual sulfur dioxide source distribution for City of Chicago, 1967.

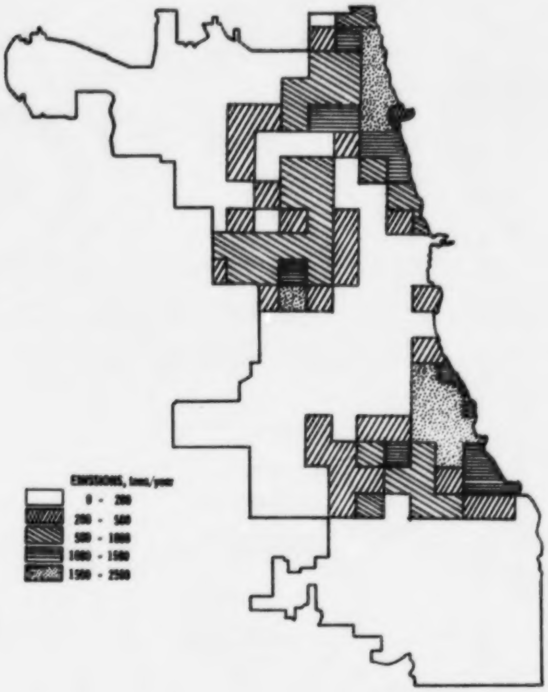

Figure 4.1.7. Sulfur dioxide emission from residential space heating, City of Chicago, 1968.

expensive; hence, it is essential to conduct studies that objectively determine the health effects of air pollutants to ensure that the cost of achieving the primary standards are not higher than necessary to adequately protect human health and welfare.

## MONITORING HUMAN EXPOSURE TO AIR POLLUTANTS

Air quality data from two monitoring networks were available in the study area:

1. City of Chicago 20-station network (City).
2. Environmental Protection Agency National Air Surveillance Network (NASN), including one Continuous Air Monitoring Program (CAMP) station.

### City

Within the metropolitan area, the City of Chicago Department of Environmental Control maintains a network of 20 air monitoring stations (Figure 4.1.1). Twenty-four-hour-average sulfur dioxide samples are obtained twice weekly and analyzed by the West-Gaeke method.[5] Total suspended particulates are obtained three times per week using the high-volume sampler.[6] The weight or mass of particulate matter per unit volume of air is determined by drawing a measured volume of air through a previously weighed clean filter and then reweighing the soiled filter.

### NASN

The NASN sampling frequency differs from that of Chicago. NASN uses 26 randomly selected days per year for both total suspended particulates and

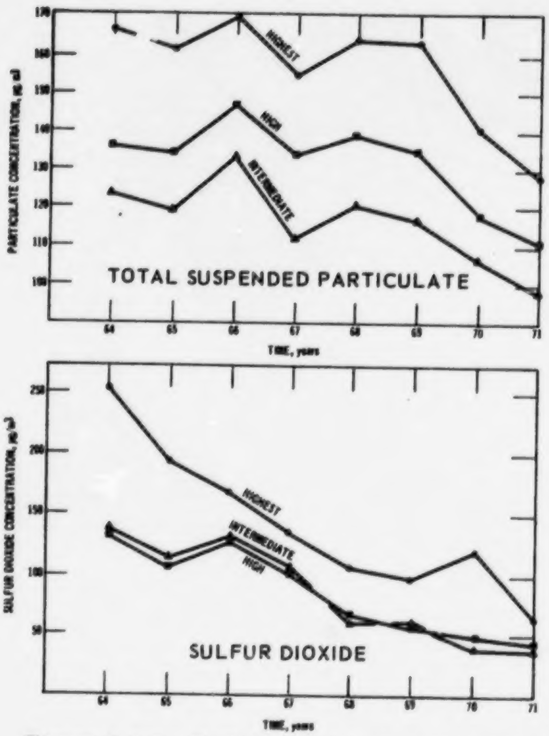

Figure 4.1.8. Pollution exposure estimates, City of Chicago, 1964-1971.

sulfur dioxide. Each sampler location, identified by station name in Figure 4.1.2, represents the central business-commercial district of that particular area. In such a location, measured concentrations are among the higher concentrations found in the immediate area. These values may, therefore, slightly overestimate area-wide exposure. In the absence of area data, however, these measurements were used as the basis for historical exposure estimates.

NASN collection procedures are similar to those of the City network for total particulates. The high-volume sampler is used to collect 24-hour samples. The suspended sulfate fraction is determined by the methylthymol blue method. Twenty-four-hour sulfur dioxide samples are analyzed using the West-Gaeke method.[5-7] Continuous measurements of sulfur dioxide concentration are made at the CAMP station in Chicago using a conductometric method.[8] The principle of this method is the oxidation of sulfur dioxide to sulfuric acid by aqueous hydrogen peroxide and the subsequent measurement of the electrical conductivity of the solution.

## RESULTS AND DISCUSSION

### Frequency Distribution

Continuous sulfur dioxide measurements for Chicago are summarized in Figure 4.1.9 in terms of cumulative frequency. These data, shown for several different years, indicate that general air quality is improving, although the National Daily Primary Standard for sulfur dioxide (365 μg/m³) was exceeded 8 percent of the days in 1970. This should be compared with a corresponding 42 percent in the 1962-1964 period.

### Exposure Estimates

Air pollution meteorological models were developed for the Chicago area by both Argonne National Laboratories and the Mitre Corporation.[9,10] These models were useful for determining the distribution of air pollutants in this area. Although both models were available for use in the study and were in general agreement for central Chicago, the Mitre model predicted a lower concentration of sulfur dioxide in the southwestern area than did the Argonne model. Measured data from the City network, from which the exposure estimates were made, were best supported by the Mitre model. An example isopleth for sulfur dioxide during 1968 based on the Mitre model is shown in Figure 4.1.10.

Exposure data are presented in two parts: first, exposures within the City of Chicago for 1964-1971, and second, exposures in the Chicago-Northwest Indiana area for 1950-1971. Data on which these estimates are based are given in the Appendix, Tables 4.1.A.1 to 4.1.A.7. Calculations performed on the data, described in the following paragraphs, are illustrated in Figures 4.1.A.1 to 4.1.A.9.

#### *City of Chicago*

In order to estimate prior exposures, data for the period 1950 through 1970 were used. Except for annual particulate dustfall in Chicago (Figure 4.1.A.1), many data points were missing. Relationships between pollutants, such as the ratio of total suspended particulates to monthly dustfall in Table 4.1.A.3, were computed to provide reasonable estimates for missing data points.

To estimate missing total suspended particulate values, an average ratio was determined from a graph of the ratio of total suspended particulates to average

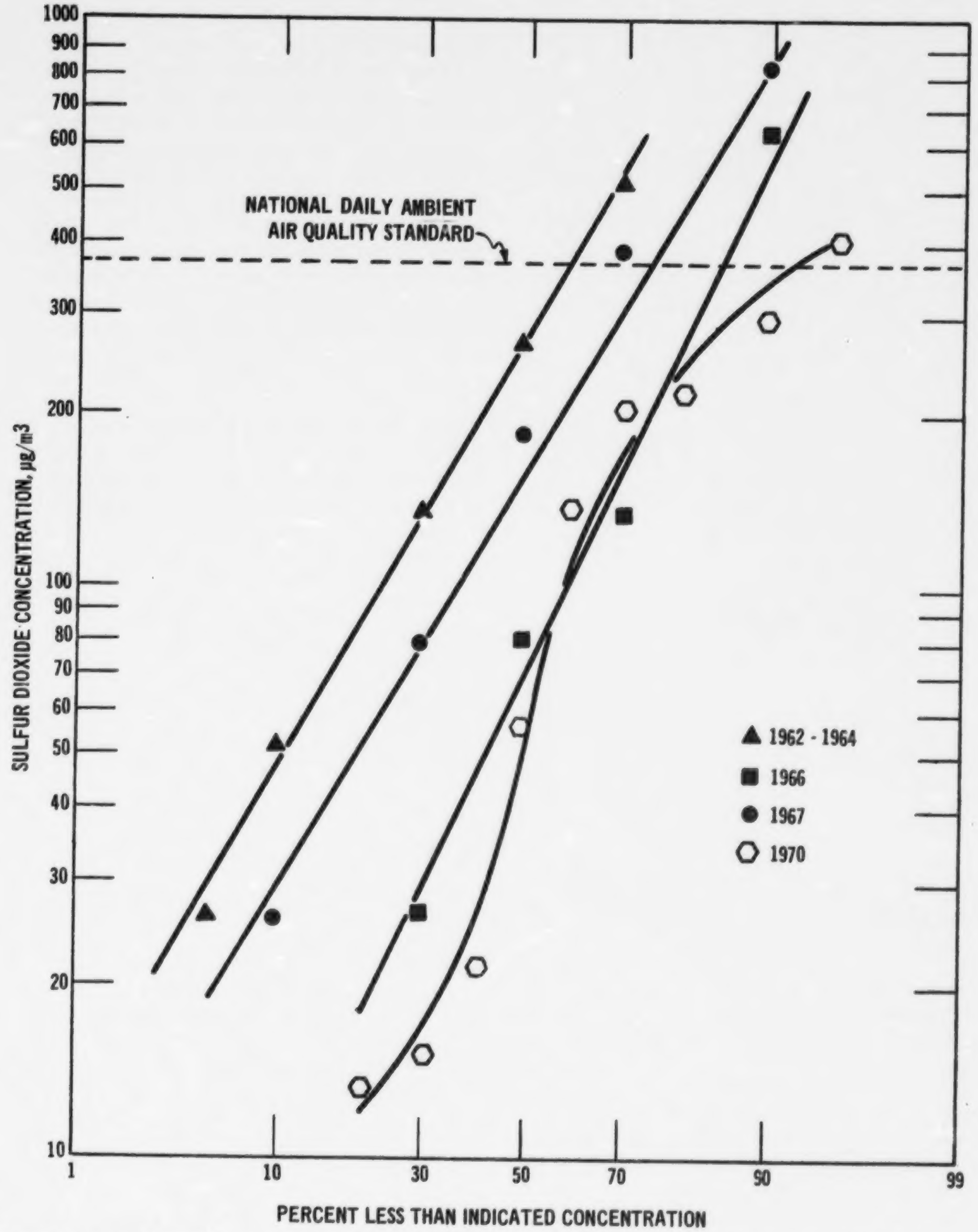

Figure 4.1.9. Cumulative frequency distribution of sulfur dioxide concentrations, City of Chicago.

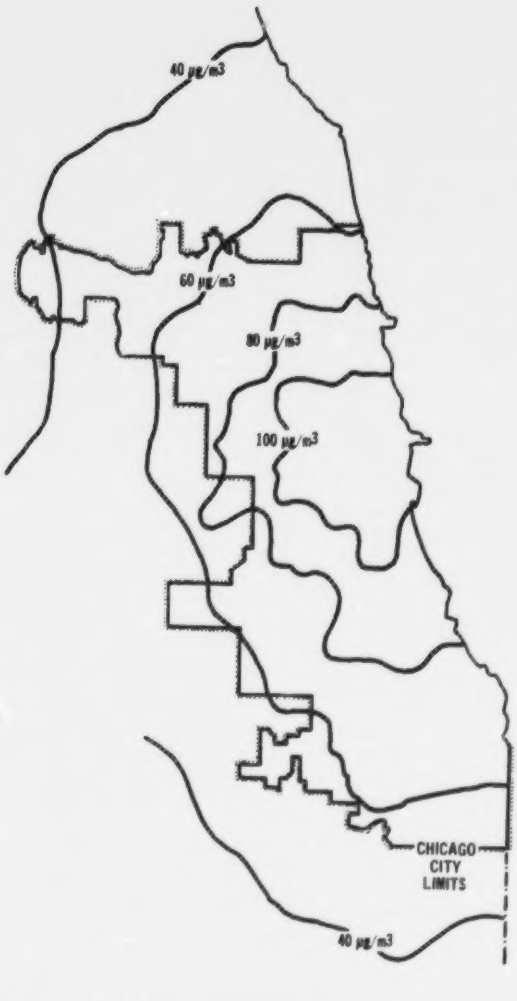

Figure 4.1.10. Sulfur dioxide isopleths for Chicago and suburbs based on 1968 emissions.

particulate dustfall versus time (Figure 4.1.A.2). Multiplication of the particulate-to-dustfall ratio by corresponding yearly dustfall values gave estimated particulate values for 1950-1953 (Figure 4.1.A.3). The correlation of total suspended particulates to dustfall is 0.75, and the regression line is plotted in Figure 4.1.A.4. Although the limitation of extrapolating to earlier years is recognized, this still provides a crude estimate for previous exposures.

Using the same approach, the ratio of suspended sulfates to dustfall was plotted versus time (Figure 4.1.A.5) and projected values were used for missing data for sulfates (Figure 4.1.A.6).

A similar comparison between the sulfur dioxide data and dustfall data showed no consistent relationship. Sulfur dioxide exposure estimates for the City of Chicago were based instead on CAMP data.

Comparison of 1967 average sulfur dioxide values from NASN Site 1 with those from the CAMP station indicated that the CAMP value was higher by 29 percent. Further comparison of CAMP values with the nearby City Station F for 1964-1969 (Table 4.1.A.1) indicated that CAMP values were approximately 15 percent higher than City values. CAMP data reduced by 25 percent are considered comparable to results from NASN Site 1. This assumption is reasonable since CAMP sampling utilizes a conductometric method instead of the West-Gaeke method and thus tends to report higher readings due to positive interference caused by other acidic gases. Data for the City of Chicago in Table 4.1.A.4 are, therefore, CAMP data reduced by 25 percent. Exposure estimates for total suspended particulates and sulfur dioxide in the City of Chicago are summarized in Table 4.1.1 for the three study areas used in the acute respiratory disease study.[1] Sulfur dioxide data for 1968 compare favorably with values predicted by the model.

### *Chicago-Northwest Indiana*

Exposure estimates for study areas used in the chronic respiratory disease study[2] were projected from recent measurements (Figures 4.1.A.7 to 4.1.A.9). Sulfur dioxide, total suspended particulate, suspended sulfate, and particulate dustfall exposures are categorized by area into Chicago-Northwest Indiana urban core (High I), suburban (High II), and relatively clean outstate (Low) in Table 4.1.2.

## SUMMARY

Sulfur dioxide, total suspended particulate, and dustfall measurements for the City of Chicago and for the Chicago-Northwest Indiana Metropolitan area have been obtained by the City of Chicago and the Environmental Protection Agency (NASN and CAMP) in recent years. Based on these measured values, it was possible to project pollutant exposure in these areas for the period 1950-1970.

The exposure estimates indicated a definite pollution exposure gradient among Chicago areas considered in an acute respiratory disease study (Intermediate, High, Highest) and between Chicago-

### Table 4.1.1. ESTIMATES OF EXPOSURE FOR CITY OF CHICAGO, 1950-1970

| Pollutant and community | Concentration, $\mu g/m^3$ | | | |
|---|---|---|---|---|
| | 1950-58[a] | 1959-63[a] | 1964-68 | 1969-70 |
| Total suspended particulate | | | | |
| Intermediate | – | 123 | 121 | 111 |
| High | – | 140 | 137 | 126 |
| Highest | 243 | 165 | 163 | 151 |
| Sulfur dioxide | | | | |
| Intermediate | – | 130 | 109 | 57 |
| High | – | 130 | 107 | 51 |
| Highest | – | 250 | 170 | 106 |

[a] Projected values.

### Table 4.1.2. ESTIMATES OF EXPOSURE FOR CHICAGO-NORTHWEST INDIANA METROPOLITAN REGION, 1950-1970

| Pollutant and community | Pollutant levels over time | | | |
|---|---|---|---|---|
| | 1950-59 | 1960-65 | 1966-68 | 1969-70 |
| Sulfur dioxide, $\mu g/m^3$ | | | | |
| Urban core (High I)[a] | – | 282 | 157 | 96 |
| Suburban (High II) | – | – | 100 | 217[c] |
| Relatively clean outstate (Low) | ~70 | ~70 | 43 | 19 |
| Total suspended particulates, $\mu g/m^3$ | | | | |
| Urban core (High I) | 244 | 177 | 149 | 155 |
| Suburban (High II) | 174[b] | 141[b] | 118 | 103 |
| Relatively clean outstate (Low) | ~80 | ~80 | 76 | 71 |
| Suspended sulfates, $\mu g/m^3$ | | | | |
| Urban core (High I) | 20.6[b] | 17.3 | 14.1 | 14.5 |
| Suburban (High II) | – | – | – | – |
| Relatively clean outstate (Low) | < 8 | <8 | 7.7 | 9.3 |
| Dustfall, $g/m^2/mo$ | | | | |
| Urban core (High I) | 19.4 | 15.2 | 14.2 | 12.6[b] |
| Suburban (High II) | – | – | – | – |
| Relatively clean outstate (Low) | – | – | – | – |

[a] Values for Chicago proper utilized.

[b] Projected values.

[c] 1969 data only.

Northwest Indiana areas considered in a chronic respiratory disease study (High I, High II, Low). In some instances, sulfur dioxide exposures in the High II area equaled or exceeded those in the High I area, possibly as a result of proximity to point sources. Low exposures for the outlying Low area, attributed to fewer sources per unit area, were verified.

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## APPENDIX

### Table 4.1.A.1. ESTIMATES OF SULFUR DIOXIDE EXPOSURE FOR CITY OF CHICAGO (CITY NETWORK)

| Community[a] | Station | Concentration, $\mu g/m^3$ | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1964 | 1365 | 1966 | 1967 | 1968 | 1969 | 1970 | 1971 | Average |
| Highest | Cooley (D) | 210 | 199 | 144 | 60 | 89 | 76 | 152 | 71 | 125 |
| | GSA (F) | 445 | 267 | 191 | 212 | 147 | 136 | 131 | 58 | 198 |
| | Crane (G) | 183 | 170 | 168 | 147 | 94 | 68 | 105 | 39 | 122 |
| | Carver (Q) | 105 | 86 | 100 | 73 | 52 | 71 | 71 | 58 | 77 |
| | Hyde Park (L) | 314 | 241 | 236 | 178 | 141 | 107 | 134 | 79 | 179 |
| High | Austin (H) | 262 | 204 | 217 | 186 | 115 | 76 | 18 | 39 | 140 |
| | Kelly (J) | 105 | 113 | 110 | 92 | 66 | 76 | 81 | 58 | 88 |
| | Chicago Voc. (O) | 105 | 52 | 86 | 66 | 39 | 13 | 18 | 16 | 49 |
| | Logan Sq. (V) | - | - | - | Data not available | | | - | - | - |
| | Clay (R) | 52 | 55 | 97 | 63 | 42 | 68 | 60 | 45 | 60 |
| Intermediate | Taft (A) | 131 | 81 | 89 | 68 | 42 | 26 | 55 | 31 | 65 |
| | Lakeview (B) | 210 | 206 | 173 | 139 | 89 | 79 | 97 | 66 | 132 |
| | Steinmetz (C) | 131 | 94 | 73 | 68 | 37 | 34 | 60 | 42 | 68 |
| | Lindbolm (K) | 131 | 121 | 131 | 105 | 79 | 60 | 56 | 34 | 90 |
| | Stevenson (M) | 52 | 66 | 115 | 110 | 71 | 39 | 18 | 29 | 63 |
| | Calumet (N) | 52 | 84 | 141 | 118 | 58 | 42 | 16 | 26 | 137 |
| | Fenger (P) | 105 | 52 | 102 | 63 | 13 | 34 | 60 | 31 | 58 |
| | Farr (I) | - | 139 | 136 | | | | | | 138 |
| | Sullivan (T) | 341 | 186 | 186 | 155 | 98 | 110 | 34 | 39 | 144 |
| | Hale (W) | 52 | 100 | 149 | 115 | 73 | 110 | 94 | 34 | 91 |

[a] Grouped according to 1969 total suspended particulate levels.

### Table 4.1.A.2. ESTIMATES OF TOTAL SUSPENDED PARTICULATE EXPOSURE FOR CITY OF CHICAGO (CITY NETWORK)

| Community[a] | Station | Concentration, $\mu g/m^3$ | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1964 | 1965 | 1966 | 1967 | 1968 | 1969 | 1970 | 1971 | Average |
| Highest | Cooley (D) | 186 | 166 | 175 | 167 | 173 | 166 | 150 | 145 | 166 |
| | GSA (F) | 178 | 173 | 174 | 172 | 161 | 160 | 134 | 125 | 160 |
| | Crane (G) | 163 | 155 | 165 | 142 | 143 | 156 | 141 | 133 | 150 |
| | Carver (Q) | 128 | 147 | 156 | 142 | 146 | 156 | 134 | 116 | 141 |
| | Hyde Park (L) | 173 | 164 | 177 | 147 | 194 | 172 | 140 | 118 | 161 |
| High | Austin (H) | 139 | 135 | 143 | 132 | 132 | 133 | 112 | 107 | 129 |
| | Kelly (J) | 146 | 132 | 147 | 132 | 137 | 133 | 115 | 106 | 131 |
| | Chicago Voc. (O) | 132 | 125 | 145 | 133 | 138 | 133 | 117 | 111 | 129 |
| | Logan (V) | 142 | 147 | 162 | 139 | 152 | 141 | 124 | 112 | 140 |
| | Clay (R) | 123 | 130 | 135 | 128 | 129 | 132 | 118 | 113 | 126 |
| Intermediate | Taft (A) | 94 | 98 | 100 | 82 | 100 | 96 | 88 | 85 | 93 |
| | Lakeview (B) | 134 | 128 | 132 | 117 | 138 | 124 | 116 | 104 | 124 |
| | Steinmetz (C) | 101 | 100 | 111 | 93 | 101 | 113 | 86 | 80 | 98 |
| | Lindblom (K) | 125 | 127 | 136 | 118 | 127 | 119 | 101 | 91 | 118 |
| | Stevenson (M) | 114 | 112 | 125 | 113 | 119 | 109 | 106 | 97 | 112 |
| | Calumet (N) | 119 | 117 | 127 | 115 | 118 | 121 | 111 | 100 | 116 |
| | Fenger (P) | 124 | 118 | 131 | 117 | 119 | 122 | 105 | 103 | 117 |
| | Farr (I) | 157 | 146 | 176 | - | 129 | 126 | 126 | 119 | 140 |
| | Sullivan (T) | 124 | 118 | 144 | 125 | 108 | 105 | 101 | 90 | 114 |
| | Hale (W) | 134 | 130 | 138 | 120 | 144 | 123 | 120 | 101 | 126 |

[a] Grouped according to 1969 total suspended particulate levels.

### Table 4.1.A.3. AIR QUALITY DATA AND RELATIONSHIPS, CITY OF CHICAGO (NASN)

| Year | Dustfall particulate, $g/m^2/mo$ | Sulfate/ dustfall ratio | Suspended sulfates, $\mu g/m^3$ | Total suspended particulates, $\mu g/m^3$ | TSP/ dustfall ratio | Sulfur dioxide, $\mu g/m^3$ |
|---|---|---|---|---|---|---|
| 1970 | 12.4[a] | | 14.8 | 124 | 10.3[a] | 72 |
| 1969 | 12.8[a] | | 14.1[a] | 172 | 10.1[a] | 184 |
| 1968 | 15.2 | 0.881 | 13.4 | 151 | 9.93 | 174 |
| 1967 | 14.6 | 0.849 | 12.4 | 143 | 9.79 | 232 |
| 1966 | 12.9 | 0.891 | 11.5 | 159 | 12.32 | 221 |
| 1965 | 14.4 | 1.10 | 15.9 | 173 | 12.01 | 341 |
| 1964 | 14.1 | 1.35 | 19.0 | 176 | 12.48 | 459 |
| 1963 | 15.8 | 1.07 | 16.9 | 144 | 9.11 | 393 |
| 1962 | 15.1 | 1.03 | 15.5 | 123 | 8.15 | 296 |
| 1961 | 17.1 | 1.46 | 24.9 | 190 | 11.11 | 350[a] |
| 1960 | 14.4 | 1.04[a] | 15.0 | 182 | 12.64 | 390[a] |
| 1959 | 17.1 | 1.05[a] | 18.0 | 240 | 14.04 | |
| 1958 | 18.3 | 0.907 | 16.6 | 190 | 10.38 | |
| 1957 | 19.1 | 1.06[a] | 20.2 | 194 | 10.16 | |
| 1956 | 20.1 | 1.21 | 24.4 | 290 | 14.43 | |
| 1955 | 18.5 | 1.03 | 19.0 | 231 | 12.49 | |
| 1954 | 19.6 | 1.05 | 20.6 | 223 | 11.38 | |
| 1953 | 18.8 | 1.08[a] | 20.3[a] | 243[a] | 12.9[a] | |
| 1952 | 19.7 | 1.09[a] | 21.5[a] | 256[a] | 13.0[a] | |
| 1951 | 22.3 | 1.10[a] | 24.5[a] | 294[a] | 13.2[a] | |
| 1950 | 20.5 | 1.11 | 22.0[a] | 273[a] | 13.3[a] | |

[a] Estimated values.

### Table 4.1.A.4. ESTIMATES OF SULFUR DIOXIDE EXPOSURE FOR CHICAGO-NORTHWEST INDIANA METROPOLITAN AREA (NASN)

| Community | Area | Concentration, $\mu g/m^3$ | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1960 | 1961 | 1962 | 1963 | 1964 | 1965 | 1966 | 1967 | 1968 | 1969 | 1970 |
| High I | Chicago[a] | 292[b] | 280[b] | 222 | 295 | 344 | 256 | 166 | 174 | 130 | 138 | 54 |
| | E. Chicago | | | | 105 | 100 | 128 | 107 | 117 | 75 | 101 | 57 |
| | Hammond | | | | | | | | | | 85 | 58 |
| High II | Harvey | | | | | | | | | 194 | 94 | |
| | Wilmette | | | | | | | | | 160 | 174 | |
| | Chicago Heights | | | | | | | 58 | 60 | 141 | 292 | |
| | Hillside | | | | | | | | | 172 | 291 | |
| | Cicero | | | | | | | | | 248 | 232 | |
| Low | Parke Co. | | | | | | 44 | | | | | 8 |
| | Monroe Co. | | | | | | | | | 10 | 14 | 9 |
| | Evansville | | | | | | | 66 | 47 | 24 | 32 | 25 |

[a] CAMP measurements times 0.75.

[b] Estimated values.

## Table 4.1.A.5. ESTIMATES OF TOTAL SUSPENDED PARTICULATE EXPOSURE FOR CHICAGO-NORTHWEST INDIANA METROPOLITAN AREA (NASN)

| Community | Area | Concentration, $\mu g/m^3$ | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | 1956 | 1957 | 1958 | 1959 | 1960 | 1961 | 1962 | 1963 | 1964 | 1965 | 1966 | 1967 | 1968 | 1969 | 1970 |
| High I | Chicago | 273[a] | 294[a] | 256[a] | 243[a] | 223 | 231 | 290 | 194 | 190 | 240 | 182 | 190 | 123 | 144 | 176 | 173 | 159 | 143 | 151 | 172 | 124 |
| | E. Chicago | | | | | | 256 | 269 | 233 | 226 | 206 | 170 | 153 | 194 | 191 | 218 | 207 | 186 | 168 | 161 | 187 | 189 |
| | Hammond | | | | | | | | | | 157 | | 132 | | 186 | | 127 | 143 | 124 | 104 | 129 | 128 |
| High II | Calumet City | | | | | | | | | | | | | | | | | 131 | 120 | 114 | 128 | 104 |
| | Harvey | | | | | | | | | | | | | | | | | 123 | 170 | 113 | 110 | |
| | Flossmer | | | | | | | | | | | | | | | | | 100[b] | 107 | 74 | 85 | |
| | Palatine | | | | | | | | | | | | | | | | | 79[b] | 74 | 80 | 80 | |
| | Wilmette | | | | | | | | | | | | | | | | | 81[b] | 139 | 78 | 72 | |
| | Blue Island | | | | | | | | | | | | | | | | | 121[b] | 145 | 130 | 119 | 78 |
| | Chicago Heights | | | | | | | | | | | | | | | | | 112[b] | 130 | 156 | 150 | 84 |
| | Hillside | | | | | | | | | | | | | | | | | 103[b] | 190 | 99 | 102 | 89 |
| | Cicero | | | | | | | | | | | | | | | | | 158[b] | 181 | 130 | 146 | 154 |
| | Franklin Park | | | | | | | | | | | | | | | | | 110[b] | 142 | 95 | 84 | |
| | Niles | | | | | | | | | | | | | | | | | 100[b] | 96 | 93 | 78 | |
| Low | Parke Co. | | | | | | | | | 58 | | 59 | 56 | 54 | 57 | 60 | 53 | 46 | 44 | 44 | 44 | 69 |
| | Monroe Co. | | | | | | | | | | | | | | | | | 48 | 51 | 42 | 42 | 36 |
| | Porter Co. | | | | | | | | | | | | | | | | 73 | | | | | 32 |
| | Rockford | | | | | | | | | | 106 | | 81 | | 95 | | 81 | 85 | 74 | 83 | 88 | |
| | Joliet | | | | | | | | | | | | | | 136 | | 132 | 118 | | 125 | 130 | |
| | Evansville | | | | | | | | 97 | | | 135 | | 86 | | 102 | 65 | | | 102 | 95 | 88 |
| | Muncie | | | | | | | | | | | | | | 99 | | | 98 | 91 | | | |
| | Springfield | | | | | | | | 94 | | | | | 109 | | | 94 | 93 | 90 | 68 | 81 | 84 |

[a] Projected values.

[b] Small sample size.

## Table 4.1.A.6. ESTIMATES OF SUSPENDED SULFATE EXPOSURE FOR CHICAGO-NORTHWEST INDIANA METROPOLITAN AREA (NASN)

| Community | Area | Concentration, μg/m3 | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1950 | 1951 | 1952 | 1953 | 1954 | 1955 | 1956 | 1957 | 1958 | 1959 | 1960 | 1961 | 1962 | 1963 | 1964 | 1965 | 1966 | 1967 | 1968 | 1969 | 1970 |
| High I | Chicago | 22.8[a] | 24.5[a] | 21.5[a] | 20.3[a] | 20.6 | 19.0 | 24.4 | 20.2[a] | 16.6 | 18.0[a] | 15.0[a] | 24.9 | 15.5 | 16.9 | 19.0 | 15.9 | 11.5 | 12.4 | 13.4 | 14.1[a] | 14.8 |
| | E. Chicago | | | | | | 21.1[b] | 21.1[b] | | 19.0 | | | | | 19.1 | 18.8 | 17.4 | 17.0 | 15.8 | 15.7 | | 16.3 |
| | Hammond | | | | | | | | | | 12.5 | | 16.7 | | 18.5 | | 13.6 | 16.5 | 12.7 | 12.3 | | 13.5 |
| Low | Parke Co. | | | | | | | | | | | | | 9.2 | | | 8.0 | 7.9 | 5.5 | 6.9 | | 13.5 |
| | Monroe Co. | | | | | | | | | | | | | | | | | 6.8 | 6.2 | 9.7 | | 7.5 |
| | Rockford | | | | | | | | | | 6.6 | | | | | | 7.5 | | 8.0 | 8.3 | | |
| | Evansville | | | | | | | | | | | | | | | 8.4 | | | | | | 9.7 |
| | Muncie | | | | | | | | | | | | | | 7.6 | | | 10.5 | 7.9 | | | |
| | Springfield | | | | | | | | | | | | | | | | 7.8 | | 7.0 | 7.0 | | 8.5 |

[a] Projected based on suspended sulfate/dustfall versus time.

[b] Average value for both years.

**Table 4.1.A.7. DUSTFALL PARTICULATE MEASUREMENTS FOR CHICAGO NASN STATION**

| Year | Dustfall, $g/m^2/mo$ | Year | Dustfall, $g/m^2/mo$ | Year | Dustfall, $g/m^2/mo$ |
|---|---|---|---|---|---|
| 1950 | 20.5 | 1957 | 19.1 | 1964 | 14.1 |
| 1951 | 22.3 | 1958 | 18.3 | 1965 | 14.4 |
| 1952 | 19.7 | 1959 | 17.1 | 1966 | 12.9 |
| 1953 | 18.8 | 1960 | 14.4 | 1967 | 14.6 |
| 1954 | 19.6 | 1961 | 17.1 | 1968 | 15.2 |
| 1955 | 18.5 | 1962 | 15.1 | 1969 | 12.8[a] |
| 1956 | 20.1 | 1963 | 15.8 | 1970 | 12.4[a] |

[a] Estimated values.

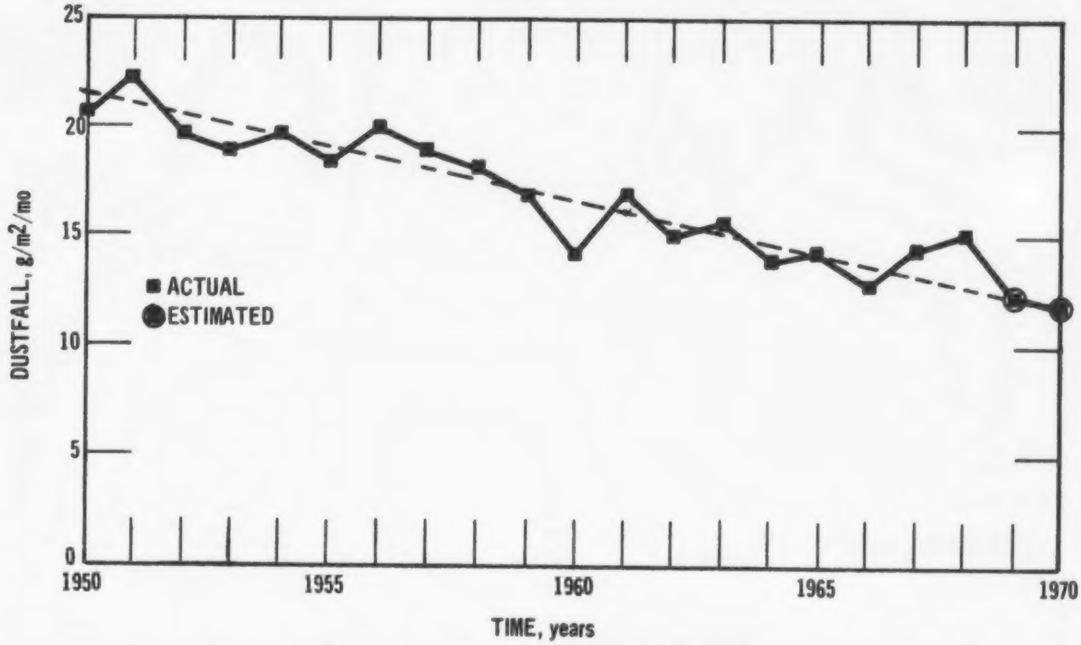

Figure 4.1.A.1. Dustfall particulate versus time, City of Chicago, 1950-1970.

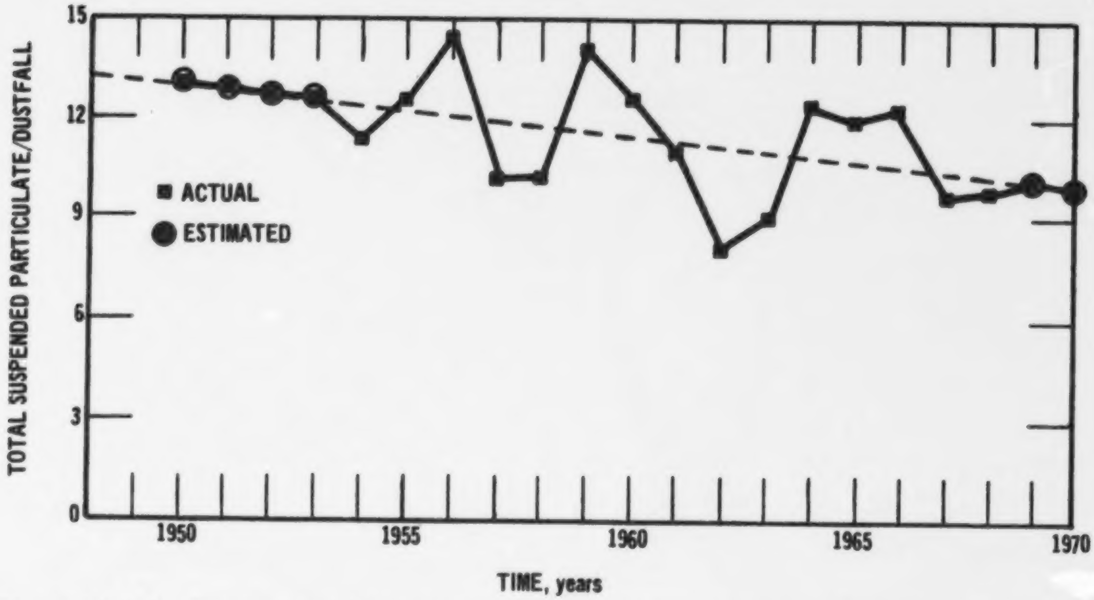

Figure 4.1.A.2. Total suspended particulate/dustfall particulate versus time, City of Chicago, 1950-1970.

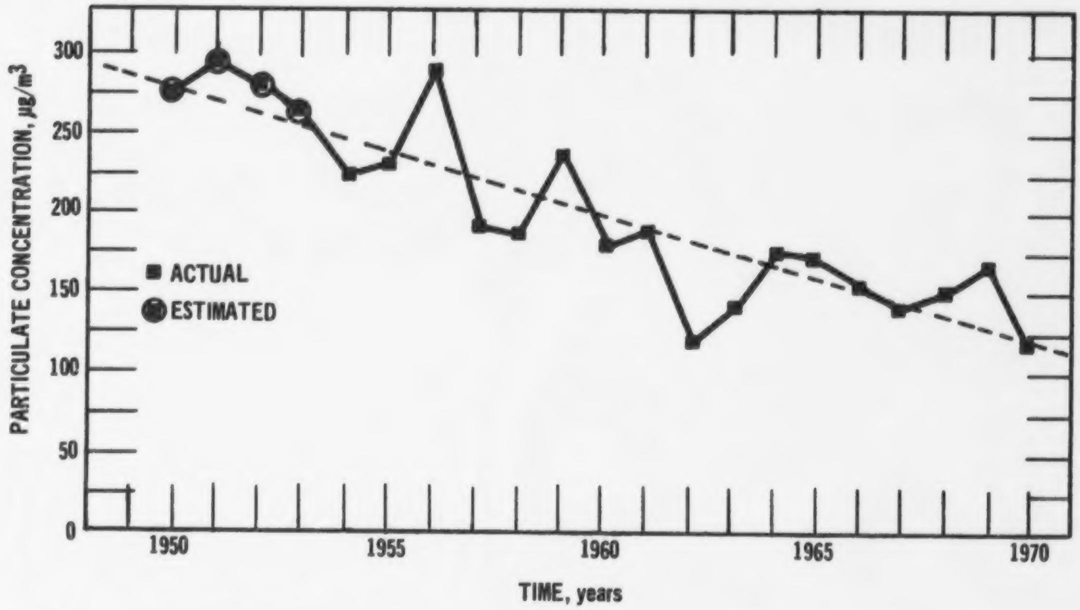

Figure 4.1.A.3. Total suspended particulate versus time, City of Chicago, 1950-1970.

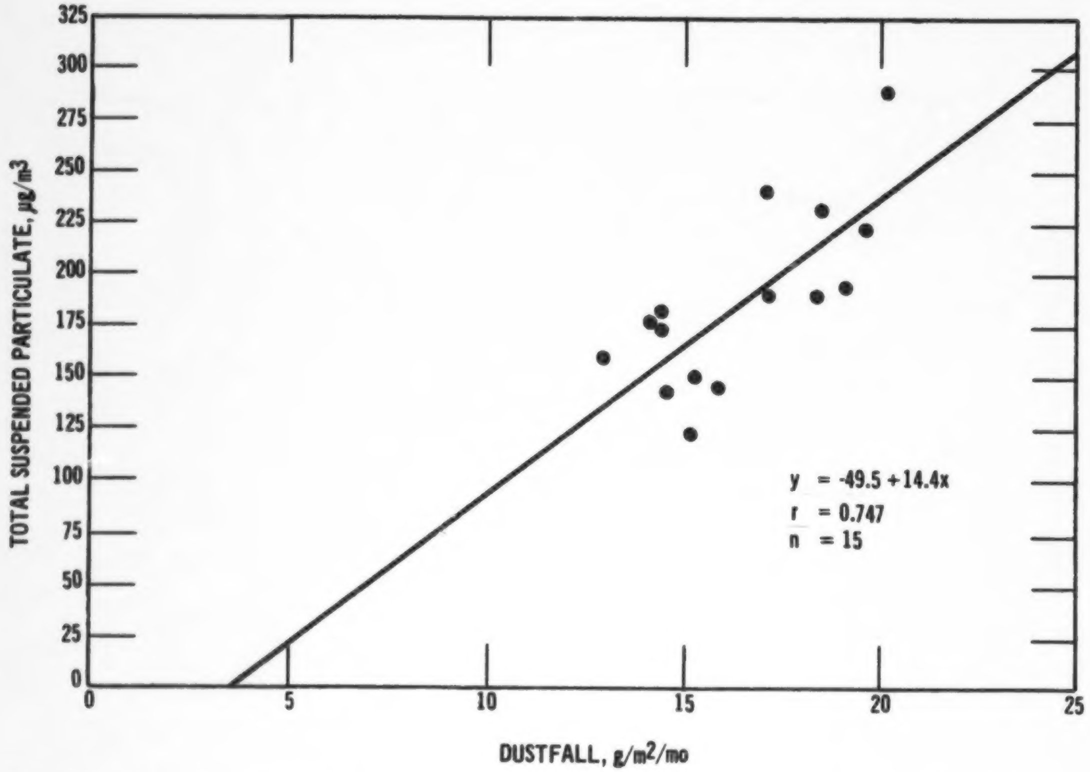

Figure 4.1.A.4. Least squares fit, total suspended particulate versus dustfall, City of Chicago, 1954-1968.

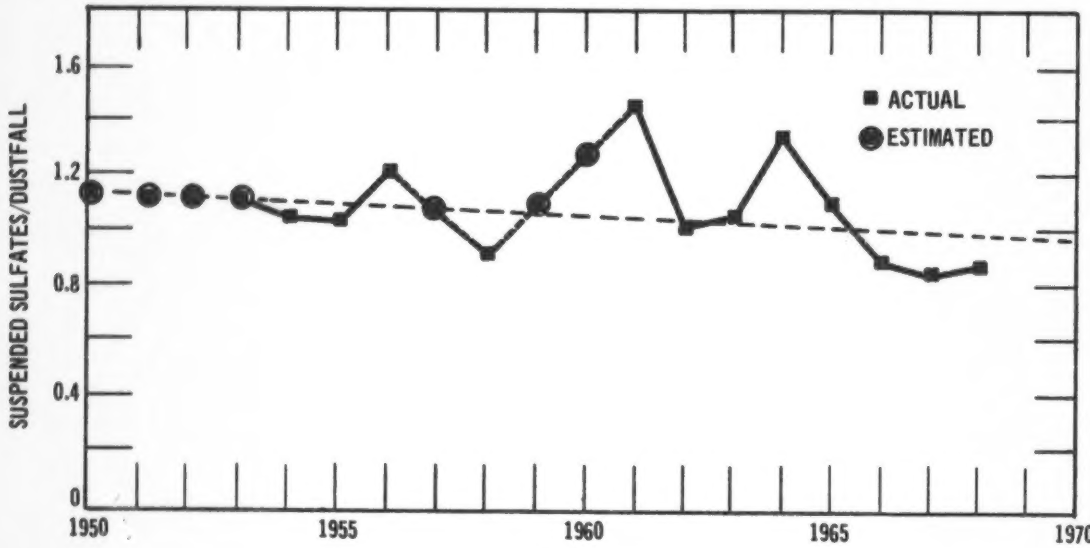

Figure 4.1.A.5. Suspended sulfate/dustfall particulate versus time, City of Chicago, 1950-1970.

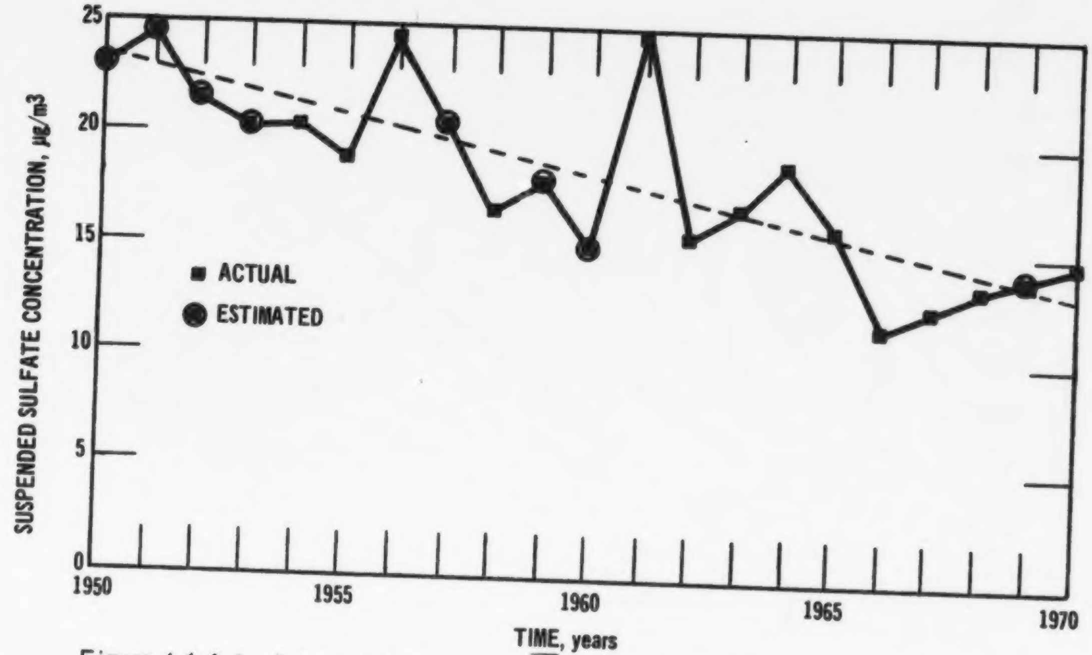

Figure 4.1.A.6. Suspended sulfates versus time, City of Chicago, 1950-1970.

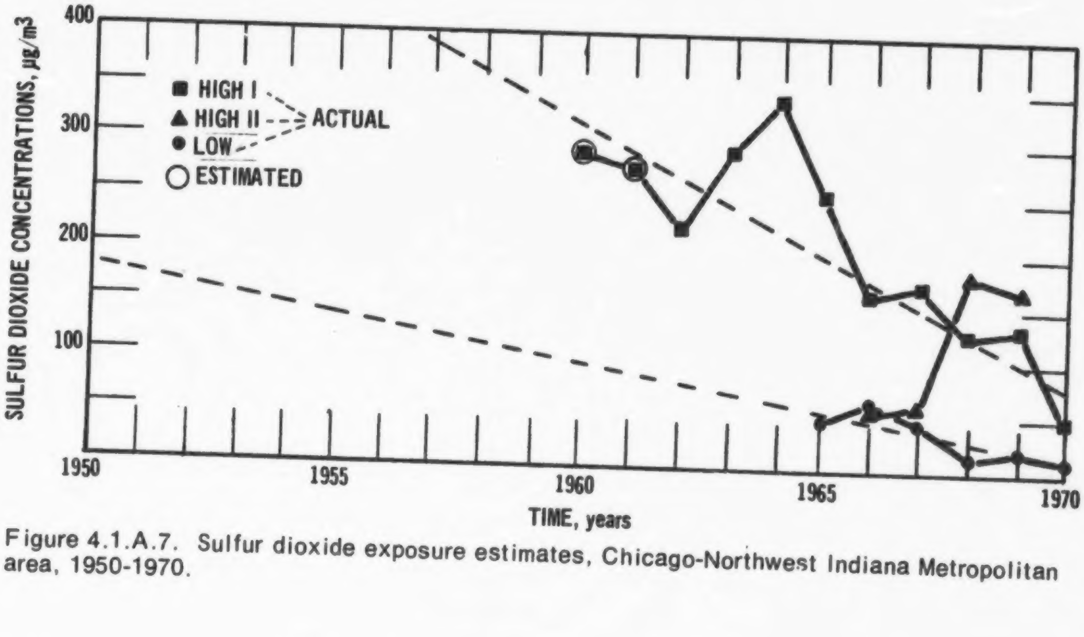

Figure 4.1.A.7. Sulfur dioxide exposure estimates, Chicago-Northwest Indiana Metropolitan area, 1950-1970.

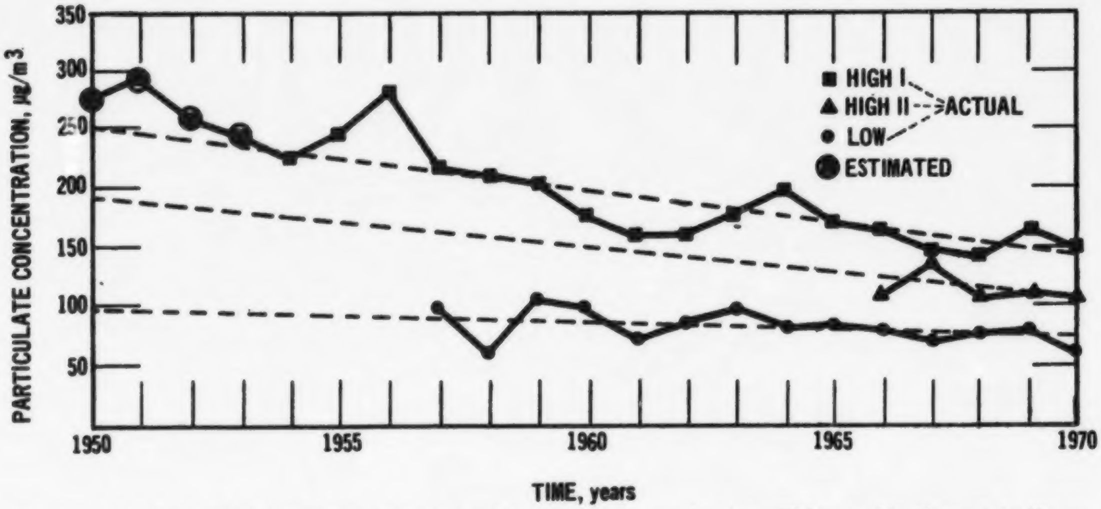

Figure 4.1.A.8. Total suspended particulate exposure extimates, Chicago-Northwest Indiana Metropolitan area, 1950-1970.

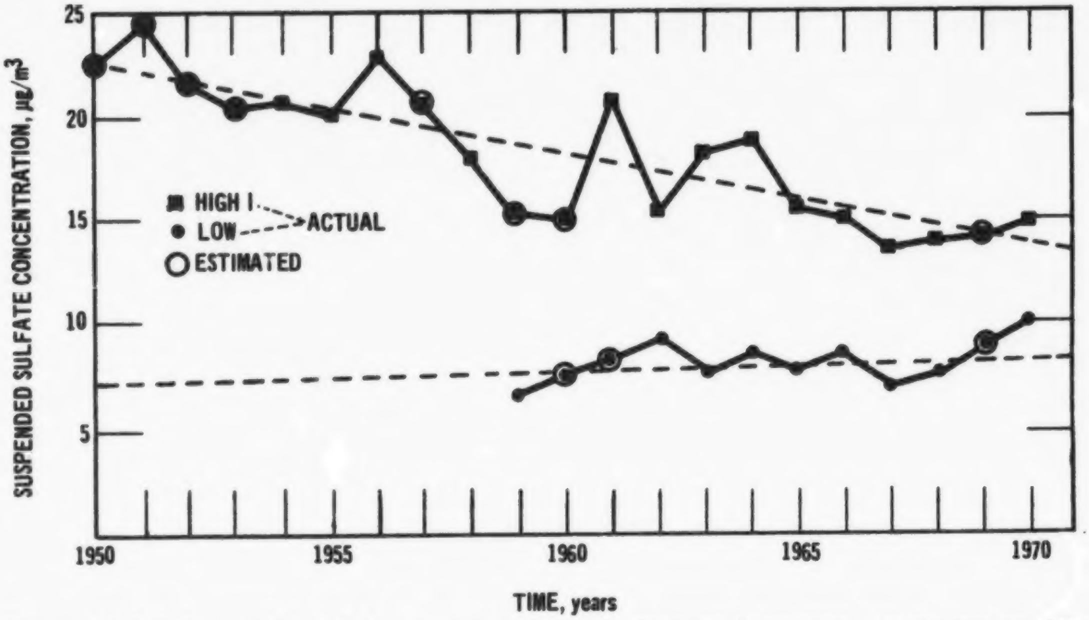

Figure 4.1.A.9. Suspended sulfate exposure estimates, Chicago-Northwest Indiana Metropolitan area, 1950-1970.

# 4.2 PREVALENCE OF CHRONIC RESPIRATORY DISEASE SYMPTOMS IN MILITARY RECRUITS: CHICAGO INDUCTION CENTER, 1969-1970

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## INTRODUCTION

This study represents an attempt to link chronic bronchitis to long-term air pollutant exposure in a population of adolescents and young adults. Self-pollution by cigarette smoke is consistently accompanied by a significant decrease in ventilatory function and a clear increase in the risk of chronic bronchitis among adults of both sexes.[1-10] In older adults, exposures to ambient air pollution are likewise accompanied by reductions in ventilatory function and increases in the prevalence of chronic bronchitis.[11-15] In late adolescence, exposures to elevated levels of air pollution perceptibly reduce ventilatory function, but the significance of this finding in contributing to the development of chronic respiratory disease symptoms in later life is still unknown.[8]

Studies involving adolescents and young adults have the advantage of allowing the assessment of relatively long-term air pollution exposures while weakening the effects of several intervening variables that hamper such studies in older adults. Selection of adolescents minimizes complex effects introduced by residential mobility, long-term self-pollution by cigarette smoking and occupational exposures involving pulmonary irritants. Unfortunately, studies of chronic respiratory disease symptoms and ambient air pollution exposures in young adults are hampered by two major difficulties: first, large populations are required to quantify the relatively small differences in symptoms that would be expected so early in life; and second, securing the cooperation of free-living young adults without introducing one or more serious study biases is most difficult. Another major disadvantage is that very few geographic areas have sufficient historical air monitoring data to allow good estimates of previous air pollution exposures.

By and large, these disadvantages were alleviated for a brief period during 1967-1969. Armed conflict in Southeast Asia resulted in a rapid acceleration of the number of military recruits being processed at induction centers. There, recruits completed medical history questionnaires and underwent physical examinations. With the cooperation of the Department of Defense, it was a relatively easy task to supplement the standard military medical history with a questionnaire dealing specifically with chronic respiratory disease symptoms. The Chicago Induction Center processes recruits from the more polluted Chicago-Northwest Indiana metropolitan urban core, from the suburbs that surround that core, and from relatively clean outstate areas of Illinois and Indiana. Of equal importance, the City of Chicago and other governmental units in the metropolitan area have maintained one or more air monitoring stations with varying degrees of sophistication since 1926.

Thus, it was possible to obtain a large sample of black and white male adults who were selected in the same fashion from more polluted and relatively clean communities. In no case did the recruits realize they were participating in a study of impact of air pollution. There was also ample opportunity to collect blood samples that may later relate immunologic and genetic risk factors to chronic respiratory disease symptom prevalence. Furthermore, linking the chronic respiratory disease survey to military records provides an opportunity for lifetime follow-up to assess the impact of the early onset of chronic respiratory disease symptoms on changes in smoking habits, on occupational choices, and on residential preferences. Currently, our understanding of the natural history of chronic respiratory disease is clouded by failure to quantify the effects of these variables.

Three specific hypotheses were tested in the study now reported: first, that residence in high pollution areas is accompanied by increases in the prevalence and/or severity of chronic respiratory disease symptoms; second, that the prevalence of chronic bronchitis is related to the length of residence in polluted areas; and third, that air pollution and cigarette smoking together have a greater adverse effect than either alone. Although other factors could have significant effects, it is difficult to perceive how they could affect the different areas in different manners.

Stringent environmental control measures began to improve air quality during the late sixties. In the future, we hope to document that the prevalence and/or severity of chronic respiratory disease is lessened by continued improvements in ambient air quality.

## METHODS

### Health Questionnaire

During the period from June 24, 1969, to February 20, 1970, all military recruits undergoing physical examination at the Chicago Induction Center received a brief, self-administered questionnaire[16] appended to the routine military medical history form. In addition to standard questions regarding age and race, six questions that dealt specifically with chronic respiratory disease symptomatology were asked. The remainder of the questionnaire pertained to smoking patterns, residential history, and educational attainment. Participants were not informed that responses would be used in a study of air pollution.

### Morbidity Indices

Frequency and duration of cough and phlegm production were used to classify each respondent on the following scale of symptom severity:

1. No symptoms.
2. Cough alone for less than 3 months each year.
3. Phlegm with or without cough for less than 3 months each year.
4. Cough alone for 3 months or more each year.
5. Phlegm alone for 3 months or more each year.
6. Cough and phlegm for 3 months or more each year.

With modification for self-administration, respiratory symptom questions were similar in scope and wording to those developed by the British Medical Research Council, and the sixth ranking on the symptom severity scales was defined as chronic bronchitis for the purpose of the study. Three summary morbidity indices were developed from these severity rankings: first, chronic bronchitis prevalence rates; second, mean symptom scores, which reflect the frequency of all reported symptoms weighted by their severity; and third, mean severity scores for those persons who reported one or more chronic respiratory disease symptoms.

### Community Pollution Exposures

Air quality for areas in and around Chicago was projected from monitoring data from the National Air Surveillance Networks, the City of Chicago, and other local and state air pollution agencies. The great majority of those recruits from outside Chicago and its suburbs came from small towns or rural areas where they were exposed to low, almost background, levels of pollution. A small proportion resided in somewhat polluted outstate industrial areas, but the paucity of aerometric measurements in these areas made it difficult to quantify pollution exposures for this population segment.

Using reported residential histories and known or estimated pollution levels, subjects were assigned to one of three exposure communities: relatively clean outstate areas, Chicago suburbs, or the urban core of the Chicago-Northwest Indiana conurbation. A small group (<3 percent) of recruits were from states other than Illinois and Indiana, and these were excluded from the analyses.

### Statistical Analyses

Comparisons were made across the three communities for the distribution of known or suspected covariates and adjustments or controls were employed where appropriate. Hypotheses were tested in a general linear model for categorical data.[17] This procedure is very similar to the analysis of variance procedures used for continuous data, except that sums of squares are replaced by Chi Squares. All rates were adjusted for educational differences, and the rates for smokers were adjusted for differences in the amount smoked.

### Environmental Exposures

Air pollution exposures were averaged over four time periods, which reflected either growth and development milestones of the study population or major shifts in air quality (Table 4.2.1).[18] The first period, 1950-1959, coincided with infancy and childhood phases of growth and development. The second period, 1960-1965, covered puberty and adolescence. The third period related to late adolescence, which also marked the onset of improvements in air quality for the City of Chicago, and the induction study years of 1969-1970 constituted the final period. During the 1950-1968 period, residents of the urban core area were exposed to extremely high levels of sulfur dioxide, suspended particulates, and suspended sulfates. Based on the limited available data, the annual average sulfur dioxide levels were estimated to be 157 to 282 $\mu g/m^3$, and the total suspended particulate levels to be 149 to 244 $\mu g/m^3$, which is 2 to 3 times the relevant National Primary Air Quality Standards.

Residents of suburban areas were perhaps more fortunate, but they were also exposed to excessive pollutant levels: estimated annual concentrations of sulfur dioxide (approximately 100 $\mu g/m^3$) and suspended particulates (118-174 $\mu g/m^3$) both exceeded the present relevant National Primary Air Quality Standards. On the other hand, residents of outstate areas enjoyed better, but probably quite variable, air quality: estimates for sulfur dioxide exposures were generally below the National Primary Air Quality

**Table 4.2.1. ESTIMATES OF EXPOSURE FOR CHICAGO-NORTHWEST INDIANA METROPOLITAN REGION, 1950-1970**

| Pollutant and community | Pollutant levels over time | | | |
|---|---|---|---|---|
| | 1950-59 | 1960-65 | 1966-68 | 1969-70 |
| Sulfur dioxide, $\mu g/m^3$ | | | | |
| Urban core[a] | – | 282 | 157 | 96 |
| Suburban | – | – | 100 | 217[c] |
| Relatively clean outstate | ~70 | ~70 | 43 | 19 |
| Total suspended particulates, $\mu g/m^3$ | | | | |
| Urban core | 244 | 177 | 149 | 155 |
| Suburban | 174[b] | 141[b] | 118 | 103 |
| Relatively clean outstate | ~80 | ~80 | 76 | 71 |
| Suspended sulfates, $\mu g/m^3$ | | | | |
| Urban core | 20.6[b] | 17.3 | 14.1 | 14.5 |
| Suburban | – | – | – | – |
| Relatively clean outstate | < 8 | < 8 | 7.7 | 9.3 |
| Dustfall, $g/m^2/mo$ | | | | |
| Urban core | 19.4 | 15.2 | 14.2 | 12.6[b] |
| Suburban | – | – | – | – |
| Relatively clean outstate | – | – | – | – |

[a] Values for Chicago proper utilized.

[b] Projected values.

[c] 1969 data only.

Standard, while particulate exposures, including a substantial agricultural background component, were somewhat below the relevant national standard.

Since 1965, sulfur dioxide and particulate levels in the urban core have been substantially reduced. By 1971, City of Chicago monitoring data indicated sulfur dioxide levels were at or below the primary standard. In 1971, suspended particulate levels (97 $\mu g/m^3$) were still somewhat above the National Primary Air Quality Standard, even though they were only half the levels monitored 10 years earlier. Air monitoring data from suburban areas, however, did not indicate the same dramatic improvement trend.

## Characterization of the Study Population

Two-thirds of the 52,000 recruits were less than 21 years of age and only 5 percent were older than 24. Tests for intercommunity differences were limited to the 45,000 recruits who completed all portions of the questionnaire. To quantify any intercommunity bias caused by these exclusions, the stepwise selection process was summarized for whites and blacks (Tables 4.2.2 and 4.2.3). The largest complete exclusion, roughly 15 percent of the population, was caused by reluctance of the respondents to record ethnic information. Only roughly 5 percent

### Table 4.2.2. WHITE RECRUITS INCLUDED IN AND EXCLUDED FROM PRINCIPAL ANALYSIS DISTRIBUTED BY COMMUNITY AND REASON FOR EXCLUSION[a]

| Community | Overall sample size | Percent excluded because of missing ethnic data | Sample size | Percentage of white recruits | | | |
|---|---|---|---|---|---|---|---|
| | | | | With complete data | | Excluded because of missing data | |
| | | | | Included | Partially excluded because of short residence | Smoking | Chronic respiratory disease symptoms |
| Relatively clean outstate | 18,864 | 13 | 16,436[b] | 62 | 32 | 1 | 5 |
| Suburbs | 12,773 | 12 | 11,298 | 66 | 29 | 1 | 4 |
| Urban core | 13,769 | 19 | 11,057 | 63 | 31 | 1 | 6 |
| Overall | 45,406 | 15 | 38,791 | 63 | 31 | 1 | 5 |

[a] Not included in the analyses are 19 recruits whose address was not listed and 126 members of miscellaneous racial groups.

[b] Not included in the analyses are 2387 recruits from areas other than Illinois and Indiana.

### Table 4.2.3. BLACK RECRUITS INCLUDED IN AND EXCLUDED FROM PRINCIPAL ANALYSIS DISTRIBUTED BY COMMUNITY EXPOSURE AND REASON FOR EXCLUSION

| Community | Sample size | Percentage of black recruits | | | |
|---|---|---|---|---|---|
| | | With complete data | | Excluded because of missing data | |
| | | Included | Partially excluded because of short residence | Smoking | Chronic respiratory disease symptoms |
| Relatively clean outstate | 871 | 46 | 38 | 3 | 13 |
| Suburbs | 441 | 50 | 35 | 3 | 13 |
| Urban core | 5422 | 48 | 34 | 3 | 14 |
| Overall | 6734 | 48 | 35 | 3 | 14 |

of the remaining white recruits and 15 percent of black recruits were excluded because of failure to answer questions on smoking habits or chronic respiratory disease symptoms. The remaining recruits were then divided into two groups: those who reported no change of address during the preceding 3 years and those who reported a recent move. Analyses of overall intercommunity differences in indices of chronic respiratory morbidity were restricted to the more stable group, while the analysis that sought an exposure duration threshold utilized both stable residents and newcomers. Black recruits appeared somewhat more mobile than white recruits. Overall, intercommunity exclusions were remarkably similar.

Intercommunity patterns in smoking history and educational attainment were then considered for each of the three exposure communities (Tables 4.2.4 and 4.2.5). There was a trend towards increased smoking in recruits of both races who lived in urban core areas. The excess in smokers was particularly striking among urban blacks (Table 4.2.4). Urban residents, whether smokers or nonsmokers, were also less likely to have completed high school than suburban or outstate recruits. Interestingly enough, this trend was closely related to smoking habits. Smokers in all communities were less likely to have completed high school, and this effect was most marked in the urban core community.

Table 4.2.4. STUDY POPULATION FOR PRINCIPAL ANALYSIS DISTRIBUTED BY COMMUNITY, RACE, AND SMOKING STATUS[a]

| Community | Blacks | | | Whites | | |
|---|---|---|---|---|---|---|
| | Percent nonsmokers | Percent smokers | Total | Percent nonsmokers | Percent smokers | Total |
| Relatively clean outstate | 34.5 | 65.5 | 789 | 38.6 | 61.4 | 10,713 |
| Suburbs | 28.5 | 71.5 | 411 | 36.9 | 63.1 | 7,676 |
| Urban core | 10.4 | 89.6 | 4,169 | 36.1 | 63.9 | 7,390 |
| Total | 15.3 | 84.7 | 5,369 | 37.4 | 62.6 | 26,779 |

[a] Limited to those living at same address for ≥3 years.

Table 4.2.5. EDUCATIONAL ATTAINMENT OF STUDY POPULATION DISTRIBUTED BY COMMUNITY AND SMOKING STATUS

| Community | Percent who completed high school | | | |
|---|---|---|---|---|
| | Blacks | | Whites | |
| | Nonsmokers | Smokers | Nonsmokers | Smokers |
| Relatively clean outstate | 80.9 | 59.2 | 93.4 | 82.3 |
| Suburbs | 77.8 | 52.4 | 94.7 | 83.0 |
| Urban core | 75.0 | 47.6 | 89.2 | 68.5 |

## HEALTH EVALUATION

### Chronic Bronchitis

Prevalence rates for chronic bronchitis were distributed by age, and when no age trends were found in the narrow range surveyed, prevalence rates for the entire population were distributed by educational attainment and smoking status (Table 4.2.6). Recruits who only completed high school were found to have somewhat lower chronic bronchitis rates than those who either did not finish high school or went beyond high school. As expected, cigarette smoking was related to chronic bronchitis prevalence in a dose-response fashion. Heavier smokers (>1 pack daily) reported rates 5 times those found in nonsmokers. Exsmokers, pipe smokers, and cigar smokers had rates lower than cigarette smokers, but higher than lifetime nonsmokers.

Residents of the more polluted urban core and suburban communities were found to have somewhat higher prevalence rates than residents of the rural areas (Table 4.2.7). These differences were significant in the smoking blacks and the nonsmoking whites (Table 4.2.8). The difference in smoking whites was nearly significant, the difference being due to a higher rate in the suburbs. The pattern among the nonsmoking blacks was mixed and not significant.

Table 4.2.6. CHRONIC BRONCHITIS PREVALENCE DISTRIBUTED BY SMOKING STATUS AND EDUCATIONAL LEVEL

| Population characteristic | Chronic bronchitis prevalence, percent |
|---|---|
| Entire study population | 12.9 |
| Education[a] | |
| Did not complete high school | 12.7 |
| Completed high school | 12.2 |
| Education beyond high school | 13.6 |
| Smoking[b] | |
| Never smoked | 5.5 |
| Exsmokers, or current pipe or cigar smokers | 7.8 |
| Current cigarette smokers: | 18.2 |
| (<1/2 pack/day) | (8.0) |
| (1/2 to 1 pack/day) | (10.5) |
| (>1 pack/day) | (28.1) |

[a] The chronic bronchitis prevalence for nonrespondents to this question was 14.6 percent.

[b] The chronic bronchitis prevalence for nonrespondents to this question was 11.6 percent.

Table 4.2.7. CHRONIC BRONCHITIS PREVALENCE DISTRIBUTED BY COMMUNITY, RACE, AND SMOKING STATUS

| Community | Chronic bronchitis prevalence, percent | | | |
|---|---|---|---|---|
| | Blacks | | Whites | |
| | Nonsmokers | Smokers | Nonsmokers | Smokers |
| Relatively clean outstate | 9.0 (8.8) | 9.3 (8.8) | 4.3 (4.2) | 16.7 (17.6) |
| Suburbs | 9.4 (7.8) | 12.6 (12.7) | 5.5 (5.4) | 19.8 (18.8) |
| Urban core | 9.4 (9.5) | 12.9 (13.0) | 5.2 (5.4) | 18.3 (17.8) |

[a] Prevalence rates adjusted for education are given in parentheses.

Table 4.2.8. ANALYSIS OF VARIANCE FOR RESPIRATORY MORBIDITY MEASUREMENTS DISTRIBUTED BY RACE AND SMOKING STATUS, CHRONIC BRONCHITIS PREVALENCE

| Factor | Degrees of freedom | Blacks | | | | Whites | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Nonsmokers | | Smokers | | Nonsmokers | | Smokers | |
| | | $X^2$ | p | $X^2$ | p | $X^2$ | p | $X^2$ | p |
| Area | 2 | 0.50 | 0.7788 | 28.79 | <0.0001 | 10.23 | 0.0060 | 4.85 | 0.0885 |
| Smoking level | 2 | – | – | 96.61 | <0.0001 | – | – | 1298.65 | <0.0001 |
| Education | 2 | 0.45 | 0.7985 | 27.04 | <0.0001 | 1.43 | 0.4892 | 109.28 | <0.0001 |
| Fit of model | 4(20)[a] | 2.83 | 0.5866 | 28.79 | <0.0001 | 1.10 | 0.8943 | 35.24 | 0.0189 |

[a] First number for nonsmokers, second for smokers.

## Mean Respiratory Symptom and Severity Scores

As previously described, mean respiratory symptom scores for each ethnic and smoking category were compiled for each exposure community. These scores were first averaged over the total population, and then a severity score was calculated for those persons who reported symptoms.

For the total study population, mean symptom scores (Table 4.2.9) were generally lowest in the relatively clean outstate communities, intermediate in the suburbs, and highest in the urban core, which also reported the highest pollution levels during most of the study period. The only exception was among white smokers, who showed almost no differences after adjusting for educational differences. Intercommunity differences in symptom scores were

Table 4.2.9. MEAN RESPIRATORY SYMPTOM SCORE DISTRIBUTED BY COMMUNITY, RACE, AND SMOKING STATUS

| Community | Mean symptom score[a] | | | |
|---|---|---|---|---|
| | Blacks | | Whites | |
| | Nonsmokers | Smokers | Nonsmokers | Smokers |
| Relatively clean outstate | 2.30 (2.10) | 2.50 (2.47) | 1.78 (1.76) | 2.98 (2.90) |
| Suburbs | 2.11 (2.24) | 2.62 (2.61) | 1.81 (1.82) | 2.98 (2.93) |
| Urban core | 2.30 (2.34) | 2.72 (2.71) | 1.88 (1.84) | 2.90 (2.91) |

[a] Scores adjusted for education are given in parentheses.

significant for smoking blacks, and nearly significant for nonsmoking whites (Table 4.2.10). Symptom scores for the other groups were not significantly different.

Mean severity scores were computed for those who reported any chronic respiratory disease symptom (Table 4.2.11). These scores had a mixed pattern, and although there were significant differences by area (Table 4.2.12), these differences were not always consistent with the estimated pollution gradient. In addition, the test for the fit of the model was significant in two of the analyses, indicating some significant interactions.

Table 4.2.10. ANALYSIS OF VARIANCE FOR RESPIRATORY MORBIDITY MEASUREMENTS DISTRIBUTED BY RACE AND SMOKING STATUS, MEAN SYMPTOM SCORES

| Factor | Degrees of freedom | Blacks | | | | Whites | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Nonsmokers | | Smokers | | Nonsmokers | | Smokers | |
| | | $X^2$ | p | $X^2$ | p | $X^2$ | p | $X^2$ | p |
| Area | 2 | 0.12 | 0.9418 | 8.72 | 0.0128 | 4.82 | 0.0898 | 0.92 | 0.6313 |
| Smoking level | 2 | – | – | 320.91 | <0.0001 | – | – | 2325.52 | <0.0001 |
| Education | 2 | 2.10 | 0.3499 | 55.70 | <0.0001 | 20.27 | 0.0001 | 75.83 | <0.0001 |
| Fit of model | 4(20)[a] | 0.79 | 0.9398 | 24.61 | 0.2168 | 0.30 | 0.9898 | 20.80 | 0.4090 |

[a] First number for nonsmokers, second for smokers.

Table 4.2.11. MEAN SEVERITY SCORES FOR RECRUITS WITH RESPIRATORY SYMPTOMS DISTRIBUTED BY COMMUNITY, RACE, AND SMOKING STATUS

| Community | Mean severity scores[a] | | | |
|---|---|---|---|---|
| | Blacks | | Whites | |
| | Nonsmokers | Smokers | Nonsmokers | Smokers |
| Relatively clean outstate | 4.00 (4.02) | 3.66 (3.62) | 3.97 (3.99) | 4.18 (4.21) |
| Suburbs | 3.67 (3.63) | 3.94 (3.98) | 4.20 (4.18) | 4.33 (4.32) |
| Urban core | 3.94 (3.93) | 3.91 (3.91) | 4.04 (4.05) | 4.24 (4.19) |

[a] Scores adjusted for education are given in parentheses.

Table 4.2.12. ANALYSIS OF VARIANCE FOR RESPIRATORY MORBIDITY MEASUREMENTS DISTRIBUTED BY RACE AND SMOKING STATUS, MEAN SEVERITY SCORES

| Factor | Degrees of freedom | Blacks | | | | Whites | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Nonsmokers | | Smokers | | Nonsmokers | | Smokers | |
| | | $X^2$ | p | $X^2$ | p | $X^2$ | p | $X^2$ | p |
| Area | 2 | 2.99 | 0.2242 | 11.27 | 0.0036 | 12.17 | 0.0023 | 18.04 | <0.0001 |
| Smoking level | 2 | – | – | 72.54 | <0.0001 | – | – | 326.24 | <0.0001 |
| Education | 2 | 8.12 | 0.0173 | 23.58 | <0.0001 | 33.23 | <0.0001 | 265.64 | <0.0001 |
| Fit of model | 4(20)[a] | 1.11 | 0.8927 | 44.55 | 0.0013 | 2.24 | 0.6917 | 55.47 | <0.0001 |

[a] First number for nonsmokers, second for smokers.

## Length of Residence

The effect of length of exposure to community air pollution was also assessed by computing chronic bronchitis prevalence rates for selected durations of residence at the same address. Only white recruits were used in these analyses because of the large sample sizes needed for the additional categorization.

Figure 4.2.1 shows an increase in the $\leq$ 1-year residence category. Smokers from all areas showed a decrease in rates for those with an intermediate length of residence, such as 4 to 7 years. All areas

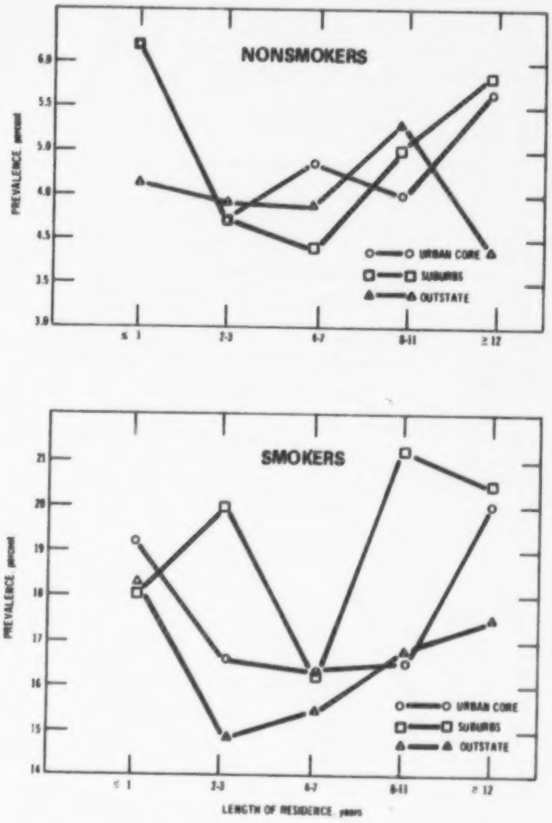

Figure 4.2.1. Bronchitis prevalence by area and length of residence. Prevalence rates for smokers adjusted for level of smoking.

showed an increase in the ≥ 12-year residence category, and the urban core and suburbs showed the greatest increase in this category. The pattern for nonsmokers was mixed, although the increase in the ≤ 1-year residence still appeared. Both the urban core and suburbs showed a sharp increase in the ≥ 12-year residence category, whereas the outstate areas did not.

Table 4.2.13 gives the relative effects of smoking and ambient air quality. These differences in prevalences were computed only for whites because of small sample sizes for blacks in some of the areas. Smokers appear to have an excess prevalence of about 13 percent, or about four times the rate for nonsmokers. Residents of the urban core and suburbs each had an excess prevalence of 1.2 percent, or approximately 1.3 times the rate for outstate areas. The combined effects of increased ambient air pollution and smoking appeared to be additive.

## DISCUSSION

Military recruits from more polluted metropolitan areas were found to have increases in two measures of chronic respiratory disease frequency. These increases were noted for smokers and nonsmokers of both major ethnic groups, although not all of the differences were statistically significant. Residence for 11 or more years in the urban core community appeared to be associated with an increase in chronic bronchitis prevalence. As expected, cigarette smoking was associated in a dose-response fashion with the prevalence of chronic bronchitis. Air pollution and cigarette smoking seemed to exert additive effects upon chronic bronchitis prevalence, although the pollution effects were not strong enough to determine this accurately.

Newcomers to an area clearly showed increases in bronchitis prevalence. This effect usually dropped off in the 2- to 3-year residence category. The effect of pollution possibly can be detected as early as 8 to 11 years, but the largest differences occur in the ≥ 12-year category. This peculiar "U" shaped distribution in the polluted areas may be partially explained by short-lived irritant symptoms induced in newcomers. The human body may then develop a tolerance to irritant effects that is not overwhelmed until exposures persist for at least 12 years. Excess morbidity among recent arrivals might also be related to a general increase in morbidity, which can in turn be related to changes in life style.[19]

The possible effects of six other intervening variables should be considered. These variables are the reliability of pollution estimates, possible effects of indoor pollutants, apparent deficit of symptoms among residents of the urban core community, socioeconomic status of families, ethnic differences within the white racial grouping, and the effect of exclusions. The procedures and rationale utilized in estimating pollutant exposures are detailed elsewhere.[18] Estimates are more reliable for the urban core than for suburban and outstate areas. Actual exposures to many residents of the urban core areas may have been somewhat lower than estimated from the available monitoring data. Existing area-wide models were available for a recent year, but they were in substantial disagreement with each other and with monitoring data from suburban stations. In general, models seemed to underestimate suburban exposures. The exposure estimates in this paper are intended as useful approximations from which to estimate health hazards; they are not precise exposure doses. In

Table 4.2.13. RELATIVE IMPORTANCE OF CIGARETTE SMOKING AND AMBIENT AIR QUALITY AS DETERMINED BY COMPARISON OF EXCESS PREVALENCE FOR CHRONIC BRONCHITIS[a]

| Current smoking status | Community | Excess prevalence of chronic bronchitis[b] | Simple additive model combining adverse effects of smoking and pollution | |
|---|---|---|---|---|
| | | | Expected | Observed/ expected |
| Nonsmokers | Outstate | 0.0 (4.2) | – | – |
| | Suburban | 1.2 | – | – |
| | Urban core | 1.2 | – | – |
| Smokers | Outstate | 13.4 | – | – |
| | Suburban | 14.6 | 14.6 | 1.00 |
| | Urban core | 13.6 | 14.6 | 0.93 |

[a] Only whites were considered since sample sizes for blacks were insufficient.

[b] Base rates are given in parentheses. Excess prevalence equals smoking and community-specific prevalence rate minus base rate.

addition, indoor levels of pollutants were not measured in the present study. Side-stream cigarette smoke and nitrogen dioxide from the use of gas for cooking or space heating may be significant indoor hazards. Domestic use of gas and cigarette smoking would be expected to be more frequent in the urban communities, but their combined effect would hardly be expected to account for the large differences attributed to ambient air pollution.

If one accepts that ambient air pollution induces or makes manifest a constitutional predisposition towards chronic respiratory disease, then higher symptom rates should have been observed in the urban core. A possible hypothesis is that ambient pollution levels in the suburbs exceeded the threshold necessary for the appearance of respiratory symptoms and that further increments in pollution would have their effects by increasing the prevalence and severity of symptoms in later life. To some extent, this speculation was supported by increases in the overall mean symptom scores for urban core residents.

Another factor that may influence symptom reporting is the socioeconomic status of the family of the recruit. Lower social status might be accompanied by real increases in disease coupled with a greater reluctance to report symptoms. The overall effect of such variation was in part controlled by consideration of racial, smoking, and educational variables. In any case, the net effect of social differences would be opposite for the poorer urban core and the more prosperous suburban communities. With this in mind, one can hardly postulate that pollution-related area differences would disappear. The effects of differences between ethnic subgroups in the study population cannot be quantitated, but they are unlikely to approach the magnitude of the reported intercommunity differences.

A more difficult problem is that of exclusions. Failure to complete the study questionnaire probably resulted in elimination of large numbers of less well educated recruits. Some estimates can be made by examining the responses of recruits who completed a portion of the questionnaire. For example, chronic bronchitis rates of those who did not classify themselves by race were identical to rates for whites and rates for those who refused to answer the smoking question were like light smokers. In other words, examination of incomplete responses revealed nothing that would diminish intercommunity differences.

After the effects of intervening variables were considered, we concluded that it would be prudent to protect the public health by assuming that increases in chronic bronchitis prevalence can be related to ambient air pollution levels characterized by annual average sulfur dioxide levels of 96 to 217 $\mu g/m^3$ accompanied by annual average suspended particulate levels of 103 to 155 $\mu g/m^3$ and annual average suspended sulfate levels of about 14 $\mu g/m^3$.

## SUMMARY

Chronic respiratory disease prevalence rates among military recruits who lived in more polluted urban core and suburban areas of Chicago were significantly higher than chronic bronchitis rates in recruits from rural and smaller urban areas in the outstate Illinois-Indiana area. Excesses in morbidity were noted among blacks, whites, smokers, and nonsmokers, although not all were statistically significant. The hazards of ambient air pollution and self-pollution by cigarette smoking seemed additive. Available evidence indicates that exposures lasting 12 years or more to ambient air pollution characterized by elevated annual average levels of sulfur dioxide (96 to 217 $\mu g/m^3$), suspended particulates (103 to 155 $\mu g/m^3$), and suspended sulfates (14 $\mu g/m^3$) were accompanied by significant increases in the frequency of chronic respiratory disease symptoms.

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# 4.3 PROSPECTIVE SURVEYS OF ACUTE RESPIRATORY DISEASE IN VOLUNTEER FAMILIES: CHICAGO NURSERY SCHOOL STUDY, 1969-1970

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## INTRODUCTION

A question of current interest is whether control of sulfur dioxide air pollution is likely to be accompanied by reductions in acute respiratory morbidity. Sulfur dioxide levels in our major urban areas are being significantly reduced by stringent, costly air pollution control measures. Acute respiratory illness is an important, multibillion dollar public health problem that is the leading cause of acute morbidity in our nation.[1,2] Substantial health benefits might result if modest decreases in the frequency or severity of acute respiratory disease were to accompany improvements in air quality. Another, possibly more important benefit is that reducing the frequency or severity of acute respiratory disease might lessen the chance of developing chronic bronchitis in later life.[3] Stringent control of ambient air pollution, cessation of self-pollution with tobacco smoke, and continued improvements in industrial hygiene offer the greatest promise for the primary prevention of chronic bronchitis.

A number of previous studies attributed excesses in acute respiratory illness to ambient air pollution characterized by elevated levels of sulfur dioxide and suspended particulates.[3-6] Increases in illnesses involving the lower respiratory tract were more consistently observed than increases in illnesses involving the upper respiratory tract. One study also linked urban pollution exposures to increased morbidity during an influenza epidemic.[7] Previous investigations have been hampered by the inability to measure pollutant exposures. All have failed to ascertain the duration of exposure needed to induce susceptibility or the persistence of susceptibility once it has been induced. This dose-response information is needed to achieve a level of environmental quality that will protect human health but not be needlessly costly.

Acute respiratory illness can best be assessed in nuclear families. Within a nuclear family, children attending nursery school manifest the highest incidence rates for acute respiratory illness.[8] Quantifying the effects of air pollution on acute respiratory disease in families and disentangling the effects of pollutants from those of age, socioeconomic status, and self-pollution by cigarette smoking is a complex task. A succession of investigations utilizing standardized techniques but separated in time and space would be needed to accumulate the mosaic of necessary information. The City of Chicago was chosen for one of the earliest studies because relatively complete air monitoring data allowed estimation of air pollution exposures and because air pollution control efforts began to achieve recognizable reductions in sulfur dioxide and total suspended particulates.

The present study sought to detect evidence of decreases in acute respiratory illness frequency or severity that might have accompanied recent reductions in ambient air pollution levels. It also attempted to ascertain which family segment was most susceptible to the deleterious effects of air pollutants as indexed by increased vulnerability to acute respiratory disease. The importance of socioeconomic status, residential mobility, and self-pollution by cigarette smoking as determinants of acute respiratory illness was assessed to assure the validity of any morbidity excess attributed to ambient air pollution.

## METHOD

### Setting and Study Population

The study population consisted of families of nursery school children aged 2 to 5 years who were attending day care centers throughout the City of Chicago. The sample was not intended to be representative of the general population but rather to provide sufficient numbers of families with both young and older children so that effects of pollution control measures could be better assessed. Middle-class residential neighborhoods such as those considered in this study do represent a large proportion of the population and are apt to be more homogeneous with respect to social class distribution. Also, neighborhood characteristics change more slowly than in central city neighborhoods.

Those day care centers selected for study were those inside Chicago city limits and situated within 1.25 miles of an air monitoring station. Letters were sent to the parent or guardian of children currently enrolled in each school requesting that the total family enroll in the study. For those who agreed to participate, a family census was obtained along with information about age of family members, crowding, education, occupation, smoking, and use of gas in the home for cooking or for heating.

### Illness Monitoring

Volunteer families were called once every 2 weeks and questioned about the presence of illness, fever, respiratory symptoms, restricted activity, middle ear infection (otitis media) diagnosed by a physician, and other physician consultation visits. A standardized questionnaire[9] was used to obtain the information, and telephone interviews were conducted by trained interviewers. After the telephone interviews were completed, all the questionnaires were reviewed for incorrect or missing information and necessary corrections were made. Whenever possible, information on symptoms was obtained from the mother; the presence or absence of illness was left to her discretion. The mother was asked whether or not any new illness had occurred in the past 2-week period. If there was an affirmative response, additional information was elicited concerning the presence of fever, restriction of activity, whether or not lower or upper respiratory symptoms were present, and whether a physician was consulted. If a member of the family had visited a physician, the informant was asked whether or not the physician had diagnosed otitis media. The upper respiratory illness classification was given to those reporting any or all of the following: cough (dry, nonproductive), head cold, sore throat, sinus or postnasal drip, or runny nose. Lower respiratory illnesses were limited to chest colds with a persistent, productive cough, bronchitis, or pneumonia. In the present study, the definition of acute lower respiratory illness was much more restrictive than that used in a later study in New York where any illness with both upper and lower tract symptoms was classed as an acute lower respiratory infection. To evaluate the validity of parental diagnosis, a small sample of parents' replies were compared with physicians' records. A total of 100 physicians were contacted concerning information on 133 individual visits.

### Air Monitoring

The air monitoring information for this study is based on measurements from 20 sampling stations located throughout the City of Chicago.[10] Total suspended particulates (TSP) were measured at each station three times a week, and sulfur dioxide was measured twice a week. Total suspended particulate levels were initially assumed to be representative of those census tracts having 20 percent or more of their area inside a circle with a radius of 1.25 miles originating at the sampling station. Monthly arithmetic mean values as well as yearly arithmetic mean values were computed for each of the areas for the year 1969. Each station was assigned to one of four quartiles (Low, Intermediate, High, Highest) based on the monitored suspended particulate levels for 1969, and analyses were made on the basis of this partitioning. Air monitoring data obtained during the 1970 period of study raises questions about the appropriateness of the groups selected, but the decision was made to proceed with the evaluation of the data on the basis of these groups. This was considered to be reasonable because during 1971 only one station from the Intermediate group and one from the High group seemed to be misassigned on the basis of sulfur dioxide levels, and these were properly assigned on the basis of total suspended particulates. Since total pollution is the factor that affects human health, any other grouping of the communities also would be questionably appropriate. As each family entered the study, it was assigned to a census tract determined by the family's residential address. If the census tract fell within the area of a monitoring station, the family was then assigned to the appropriate quartile. If the census tract did not fall within the area of a monitoring station, it was then plotted on a map of the City of Chicago to determine its proximity to the various stations. Those families in the immediate area of a station were included in the study and assigned an appropriate number. The two lower exposure quartiles were very similar with respect to 1969 pollution concentrations of total suspended particulates and sulfur dioxide. These populations were combined for the morbidity analyses to gain a larger population in the cleaner areas.

The three exposure categories grouped according to the 1969 levels of total suspended particulates were:

1. Intermediate exposure (combined Low and Intermediate quartiles), where sulfur dioxide levels were generally within the limits set by the National Secondary Air Quality Standard for sulfur dioxide and suspended particulate levels were below the median annual level for Chicago stations.
2. High exposure, where sulfur dioxide levels were within the National Secondary Air Quality Standard but suspended particulate levels were above the median level for Chicago stations and averaged 1-1/2 times the relevant National Primary Ambient Air Quality Standard.
3. Highest exposure, where sulfur dioxide levels exceeded the National Primary Ambient Air Quality Standard and suspended particulates were approximately twice the relevant National Primary Air Quality Standard.

## Statistical Analysis

The statistical analysis involved five steps. First, selected characteristics of the family groupings that comprised each exposure category were compared to detect gross socioeconomic and demographic differences that might bias the study. Second, temporal trends in mortality reporting were evaluated to isolate any epidemic outbreaks for special analysis. Third, intercommunity differences in illness frequency were assessed by relative risk models. Fourth, differences in the severity of illness were quantified and indices of excess morbidity computed. Fifth, the impact of selected intervening variables, including social status, mobility, and cigarette smoking, was evaluated.

The analytical protocol allowed partition of the entire study period into several seasonal segments so that any epidemic outbreak might be investigated separately. This procedure also minimized the adverse effects of family withdrawals or temporary nonparticipation since the experience of a family was not considered in the analysis if contact could not be made during any portion of a particular seasonal segment. Three segments were identified for the present study. The first season, which lasted from December 14, 1969, to March 21, 1970, encompassed an epidemic caused by $A_2$/Hong Kong influenza. A second, noninfluenza respiratory disease season included two periods, the early spring of 1970 and the fall of 1970, and ended just after Thanksgiving. The third (summer) segment lasted from May 31, 1970, through September 19, 1970.

When assessing illness frequency, attack rates for upper, lower, and total acute respiratory illness were computed separately for five family segments: fathers, mothers, older siblings, nursery school children, and younger siblings. The experience of other adults living in the homes was excluded from the study. To ascertain the population reporting repeated bouts of acute respiratory illness, the illness experience of each volunteer was considered. Repeated excessive illness was defined as more than three illnesses involving the upper respiratory tract, more than one illness involving the lower respiratory tract, or more than four total acute respiratory illnesses. Intercommunity differences in the frequency of acute respiratory illness were ascertained by comparing two measures: attack rates and proportion of the study population reporting excess respiratory illness.

Intercommunity differences in illness severity were ascertained by comparison of severity scores that ranged from Level I through Level V. Briefly, Level I was illness without restricted activity, Level II included restricted activity but not fever, Level III included restricted activity and fever, and Level IV included all of the preceeding plus a physician visit. Level V applied only to children and was limited to illnesses characterized by restricted activity, fever, and middle ear infection diagnosed by a physician. Level V was chosen to reflect a common, presumably bacterial, complication of acute upper respiratory illness. Levels II through V were further characterized by the mean number of restricted activity days incurred.

The primary statistical tools used in this report were the techniques developed by Grizzle et al.[11] for the analysis of categorical data. These techniques permit one to treat categorical data in a manner similar to the way continuous data are treated by the theory of least squares analysis. Since we wanted a conservative Type II error while maintaining a reasonable Type I error, a significance level of $p < 0.10$ was chosen.

## RESULTS

### Air Pollution Exposures

Health impairments attributed to air pollution might be related to either current or prior exposures and each of these could be related to either immediate or persistent impairments. Effects of recent or remote exposures might in turn be related to repeated short-term elevations of pollutants or to longer term annual average levels. Annual average air pollution exposure estimates during and for the decade prior to this study were available. However, it was not possible to evaluate recent or remote short-term peak exposures to air pollutants because constructing an aggregate frequency distribution for the multiple stations comprising each exposure stratum proved very difficult. The network of stations was designed to assess long-term exposures, and sampling was restricted to 2 to 3 days each week. There was also some unavoidable lack of comparability among monitoring data from stations within particular exposure strata because of differences in measurement techniques used through the preceeding decade. However, appropriate scale factors have been utilized to provide a common base for comparison.

Within the confines stated above, reasonable estimates of annual average air pollutant exposures could nevertheless be made for sulfur dioxide and total suspended particulates (Table 4.3.1). No matter what

### Table 4.3.1. ESTIMATED ANNUAL AVERAGE POLLUTION LEVELS IN CHICAGO STUDY COMMUNITIES

| Pollutant | Community | Concentration, $\mu g/m^3$ | | |
|---|---|---|---|---|
| | | Years prior to study | | During study (1969-70) |
| | | 6-10 (1959-63) | 1-5 (1964-68) | |
| Sulfur dioxide | Intermediate | 130 | 109 | 57 |
| | High | 130 | 107 | 51 |
| | Highest | 250 | 170 | 106 |
| Total suspended particulate | Intermediate | 123 | 121 | 111 |
| | High | 140 | 137 | 126 |
| | Highest | 165 | 163 | 151 |

exposure stratum was considered, those children who were in elementary school during the study had been exposed to excessive levels of sulfur dioxide (130 to 250 $\mu g/m^3$) and suspended particulates (123 to 165 $\mu g/m^3$) during their early years of life. Children less than six who lived in the Intermediate and High exposure communities were exposed to levels of sulfur dioxide of 107 to 109 $\mu g/m^3$ during their early childhood years. Children from the Highest exposure community were exposed to sulfur dioxide levels of 170 $\mu g/m^3$. During the study years (1969-1970), children living in the Intermediate and High exposure communities were exposed to sulfur dioxide levels (51 to 57 $\mu g/m^3$) that had fallen below the relevant National Secondary Ambient Air Quality Standard. Their counterparts in the Highest exposure community continued to be exposed to elevated levels (106 $\mu g/m^3$) of sulfur dioxide, even though these levels were appreciably reduced from concentrations of the earlier decade. Suspended particulate levels in every exposure community were highest during the earlier periods considered, and their subsequent decreases were more modest. Levels for each of the exposure communities exceeded the relevant National Primary Air Quality Standard throughout the 12-year period. There were important intercommunity differences in total suspended particulate levels that made further pooling of study populations inadvisable. During the study, suspended particulate levels in the Intermediate community (111 $\mu g/m^3$) were modestly elevated, while those in the two more polluted communities exhibited stepwise increases (126 and 151 $\mu g/m^3$).

Quantitatively less information is available for other measured ambient air pollutants. Annual average suspended sulfate levels were elevated (estimated as 18 $\mu g/m^3$ for 1959-1963, 14.4 $\mu g/m^3$ for 1964-1968, and 14.5 $\mu g/m^3$ for 1969-1970), but probably varied little between community groupings. Suspended sulfate levels appeared to decrease during the decade prior to the study. Percentagewise, this decrease in suspended sulfates resembled the fall in sulfur dioxide levels more than the gradual decline in suspended particulates. Suspended nitrate levels in the area were relatively low (1 to 2 $\mu g/m^3$ during 1960-1967).

## Characteristics of the Study Population

Intercommunity differences involving intervening variables that might require isolation or special adjustments during the principal analyses were evaluated (Table 4.3.2). Restriction of recruitment to families with children in nursery school was itself a factor that ensured a certain amount of socioeconomic homogeneity. The study subjects in each community were well educated, but a greater proportion of fathers in the Highest exposure community had attended graduate school. With the exception of a higher number of professionals in the highest pollution sector, there were no major intercommunity differences in the occupation of the fathers. In every community, the great majority of families cooked with gas, a practice that may cause elevated indoor levels of nitrogen dioxide. Cigarette smoking

Table 4.3.2. PANEL CHARACTERISTICS

| Characteristic | Percent of indicated segment by community | | |
|---|---|---|---|
| | Intermediate | High | Highest |
| Educational attainment of fathers | | | |
| High school | 92 | 90 | 94 |
| > 4 yr college | 40 | 42 | 64 |
| Families using domestic gas | 83 | 87 | 84 |
| Current cigarette smokers | | | |
| Mothers | 40 | 38 | 42 |
| Fathers | 33 | 29 | 36 |
| Families moving during past 3 yr | 46 | 44 | 56 |

seemed somewhat less frequent among parents living in the High exposure community, and families living in the Highest exposure communities reported greater residential mobility. There were essentially no intercommunity differences in smoking intensity of those with that habit. When one compared only the Intermediate and Highest communities, the only differences were in residential mobility and educational attainment. These covariates were given special attention in the analysis.

A total of 627 families representing 2705 individuals participated in the study, with the number of families participating in each community differing substantially (Table 4.3.3). The Intermediate exposure community had the most families (335) and the High area had the fewest (72). The number of families considered in the analysis is somewhat less than this due to attrition during the study.

Physicians' offices were surveyed to validate physician visits reported to the telephone interviewer. Four-fifths, or 106, of the reported visits could be identified in physician records and mother and physician concordance could be examined. For all respiratory symptoms, a 95.3 percent concordance was obtained; for upper respiratory infection, 80.2 percent; for presence of fever, 75.5 percent; and for earache, 85.8 percent. There was no reason to believe there was any difference among communities in illness reporting.

### Seasonal Variation and Epidemic Influenza

The study began November 1969 and terminated November 1970. A summer component was included to allow calculations of estimates that would be useful in evaluating similar studies that are restricted to school months, i.e., the season of highest incidence in the United States. Acute respiratory disease attack rates for each of the study communities during each reporting period were plotted to define epidemic outbreaks or serious reporting anomalies (Figure 4.3.1). Excessively high illness reporting during the initial calling period, not uncommon in panel studies, probably represented overreporting during a period when the telephone interview is viewed as a special event rather than another routine task. For this reason, data for the first three reporting periods were not included in the analysis.

One month after the study began, an epidemic wave of illness struck every community. The outbreak, subsequently associated with $A_2$/Hong Kong influenza, burned slowly through the city before subsiding in early March to baseline attack rates for the respiratory season. Thereafter, illness rates fell to seasonal lows in July and rose sharply when school resumed in September. The less polluted Intermediate community generally manifested the lowest attack rates during the respiratory seasons (influenzal and noninfluenzal), but no noteworthy intercommunity

The mother or female guardian was instructed to answer the questions for parents living in the home. Other information ascertained included length of residence in the community, educational attainment of head of household, cigarette smoking habit, age, and whether the parent had occupational exposure to respiratory irritants. It should be made clear that covariate information was applicable to the time at which the survey was conducted. For example, exsmokers were individuals who at the time of the survey were not smoking, but who had smoked in the past.

## Assessing Air Pollution Exposure

At the time of this study, air monitoring stations were located within each community. Each station was on a building rooftop at a height of about 25 feet above the ground. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high-volume samplers), suspended sulfates, and suspended nitrates were monitored daily. Daily measurements of nitrogen dioxide (Jacobs-Hochheiser method) were also made; however, the validity of the method has subsequently been questioned by the Environmental Protection Agency. Dustfall was determined from monthly samples. A full description of the monitoring station locations and aerometric procedures has been presented elsewhere.[15]

While these stations allow estimates of pollutant exposure for prospective studies, it was also necessary to estimate exposure for past time periods. Sulfur dioxide had been monitored in the Low and Intermediate I communities since 1965 and in the High community since 1970 by the Utah State Division of Health. In addition, measurements of total suspended particulates and suspended sulfates dating back to 1953 were available for the Intermediate I community. Estimates of sulfur dioxide, total suspended particulates, and suspended sulfate concentrations in the High exposure community for 1940-1970 and the Intermediate II exposure community for 1950-1970 were obtained by a mathematical dispersion model, which utilized emissions from the industrial source and extensive local meteorological data, and by observed relationships among pollutants. Observed suspended particulate, suspended sulfate, and sulfur dioxide concentrations for 1970-1971 were used to calibrate the models used to estimate exposure levels for previous years.

## Data Analysis

For the purposes of statistical analysis, a scale of symptom severity was devised by a panel of physicians based upon the number, clinical significance, and duration or persistence of symptoms. Although the number of symptom categories was somewhat arbitrary and their severity ranking was subjective and no more than ordinal in nature, the following ranking of chronic respiratory symptoms categories was developed:

1. No symptoms.
2. Cough alone for less than 3 months each year.
3. Phlegm with or without cough for less than 3 months each year.
4. Cough without phlegm for 3 months or more each year.
5. Phlegm without cough for 3 months or more each year.
6. Cough and phlegm for 3 months or more each year.
7. Cough and phlegm for 3 months or more each year and shortness of breath.

Reported respiratory symptoms were used to devise three indices of morbidity; first, chronic bronchitis was defined in accordance with the British Medical Research Council (cough and phlegm on most days for at least 3 months each year), and prevalence rates were compared across areas. The second variable was a mean respiratory symptom score defined as the arithmetic average of the respiratory symptom score of each member of a particular population group. The third variable was the arithmetic average of the respiratory symptom score for those members of the population who reported any chronic respiratory symptoms, that is, for those members who had symptom scores 2 through 7 only. The first two variables reflect frequency of chronic respiratory disease symptoms, with the mean symptom score having the advantage of considering a gradient of symptoms but the disadvantage of lacking the acceptance and the assured clinical relevance of classically defined chronic bronchitis. The mean severity score, which is the third variable, was utilized as a measure of severity of reported symptoms.

Hypotheses were tested using a general linear model for categorical data.[16] This technique uses weighted regression on categorical data and allows estimation of each effect in the model after adjusting for all other effects in the model. Hypotheses were tested by Chi Square procedures. Covariates used in

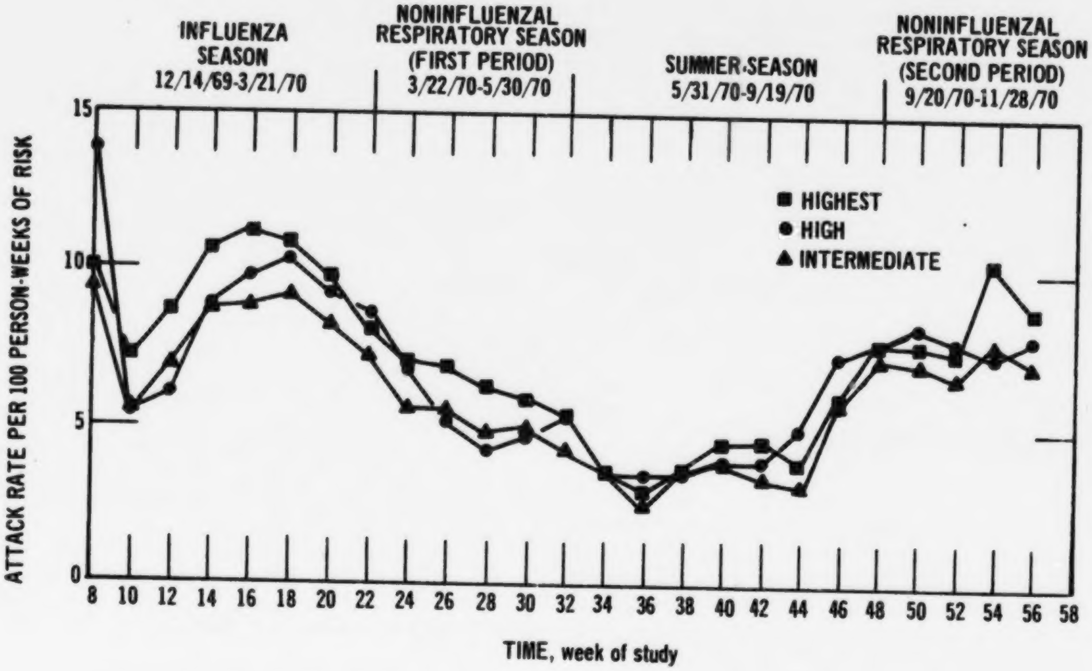

Figure 4.3.1. Acute respiratory illness attack rates versus time, December 1969 to November 1970.

reported in the Highest community. On the other hand, attack rates for upper tract illness and total acute respiratory illness were not elevated in the Highest community at that time. Since the analyses indicated a consistent pattern of variance in respiratory illness rates during both epidemic and nonepidemic periods, subsequent analyses included data from the entire study period.

### Overall Illness Frequency

For each community, overall acute respiratory disease attack rates per 100 person-weeks at risk were computed separately for each of the five family segments. Sets of relative risks were then calculated for each family segment using the Intermediate community as a base. With a longer time span, it was possible to assess the experience of the relatively limited population that comprised the High community. Even then, illness rates for this group were somewhat unstable since these rates were based upon only 850 to 1600 person-weeks of risk. Families from the Highest community consistently reported excesses in total respiratory illness and even larger excesses in lower respiratory illness, the only exception being infants, who reported no excess lower tract disease (Table 4.3.5). Rates for the High community were much more variable but tended to be higher than those in the Intermediate community. One might conclude that the more polluted groupings tended to have more total illness, and an excess in the potentially more dangerous illnesses involving the lower respiratory tract.

### Illness Severity

Each illness was assigned a severity score based upon the previously defined five-level severity scale. To facilitate intercommunity comparisons, sets of relative severity scores were calculated for each family segment. The procedure was identical to that utilized in the relative risk model except that the base was a severity score not an attack rate (Table 4.3.6). Relative severity scores were quite variable, and

### Table 4.3.4. RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS DURING $A_2$/HONG KONG INFLUENZA OUTBREAK

| Family segment | Community | Relative risk of acute respiratory illness[a] One episode | Relative risk of acute respiratory illness[a] More than one episode |
|---|---|---|---|
| Fathers | Intermediate | 1.00 (28) | 1.00 (6) |
| | Highest | 1.46 | 0.44 |
| Mothers | Intermediate | 1.00 (46) | 1.00 (12) |
| | Highest | 1.24 | 0.74 |
| Older siblings | Intermediate | 1.00 (45) | 1.00 (14) |
| | Highest | 1.20 | 1.45 |
| Nursery school children | Intermediate | 1.00 (69) | 1.00 (28) |
| | Highest | 1.07 | 0.97 |
| Younger siblings | Intermediate | 1.00 (74) | 1.00 (34) |
| | Highest | 1.08 | 1.51 |

[a] Base rate, that is percentage ill during outbreak, is in parentheses.

pollutant-attributable excesses in severity were found only for illnesses involving the lower respiratory tract in fathers and school children.

Illnesses occurring in the more polluted groupings were as severe as illnesses among families living in the less polluted Intermediate exposure area. As previously discussed, strict definition of acute lower respiratory illness was reflected in higher severity scores when contrasted to severity scores for upper tract illness. The average number of restricted activity days was also calculated for upper and lower tract illnesses of a given severity level (Table 4.3.7). Since the patterns indicated by the data were more consistent when residentially stable families were considered, the analysis in Table 4.3.7 is restricted to families that had not moved within the past 3 years. Effects of residential mobility are discussed in more detail in a later section. Increasing severity scores from Level II to Level III did not increase the number of restricted activity days in upper tract illnesses among children of any age or in lower tract illness among school children. A partial explanation might be that febrile upper tract illnesses are more often bacterial and thus more readily respond to antimicrobial therapy than do some of the more lingering infections produced by other agents.

The excess number and excess percentage of restricted activity days attributable to living in the Highest exposure community as compared with living in the Intermediate community were calculated from community-specific attack rates and severity scores for each family segment (Table 4.3.8). Since the excess days are based on 100 person-weeks of risk, a good estimate of excess restricted activity in a single year can be obtained by dividing the excess days given in Table 4.3.8 by two. (The percentages, of course, are applicable to any time frame chosen.) The annual excess is impressive and would involve about 1 extra day of restricted activity every year for each member of the family. A nuclear family with three children living in the Highest exposure community might experience as much as 12 percent more restricted activity days than a family living in the Intermediate exposure community. The greatest percentage increase in restricted activity was occasioned by acute lower respiratory illness in fathers.

There were no intercommunity differences in the percentage of illnesses requiring a physician's visit.

## Table 4.3.5. RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS FOR OVERALL STUDY PERIOD

| Family segment | Community | Relative risk of acute respiratory illness[a] Upper tract | Lower tract | Total |
|---|---|---|---|---|
| Fathers | Intermediate | 1.00 (2.66) | 1.00 (0.44) | 1.00 (3.09) |
| | High | 0.96 | 0.89 | 0.95 |
| | Highest | 1.14 | 1.48 | 1.19 |
| Mothers | Intermediate | 1.00 (4.28) | 1.00 (0.72) | 1.00 (5.00) |
| | High | 1.21 | 1.08 | 1.19 |
| | Highest | 1.14 | 1.46 | 1.19 |
| Older siblings | Intermediate | 1.00 (4.03) | 1.00 (0.53) | 1.00 (4.56) |
| | High | 1.36 | 1.64 | 1.39 |
| | Highest | 1.02 | 1.32 | 1.06 |
| Nursery school children | Intermediate | 1.00 (7.70) | 1.00 (1.67) | 1.00 (9.37) |
| | High | 1.09 | 0.92 | 1.06 |
| | Highest | 1.02 | 1.38 | 1.09 |
| Younger siblings | Intermediate | 1.00 (7.87) | 1.00 (2.87) | 1.00 (10.31) |
| | High | 1.12 | 0.37 | 0.94 |
| | Highest | 1.21 | 0.94 | 1.16 |

[a] Base rate per 100 person-weeks of risk is in parentheses.

However, physician visits for otitis media complicating an acute respiratory illness were elevated among children younger than age six who lived in the most polluted community (Table 4.3.9).

## Vulnerability to Repeated Acute Respiratory Illness

During the study, family members who reported either four total respiratory illnesses or more than three upper or one lower illness were judged to have repeated excessive illness. When the illness experience of residentially stable families in the study communities was reviewed, excessive repeated total acute respiratory illness was consistently more common in families from the Highest community (Table 4.3.10).

## Validity of Analyses

Several analyses were performed to determine whether intervening variables rather than differences in air pollution levels could account for the observed intercommunity difference in morbidity. Intervening variables considered included socioeconomic status (as indexed by educational attainment), residential mobility, and cigarette smoking.

Since residential mobility and educational attainment appeared to be related to each other, the effects of educational attainment were first evaluated for the entire study population and later evaluated for residentially stable families only. To evaluate educational differences, attack rates for each type of acute respiratory illness were adjusted for the educational attainment of fathers using a five-level educational achievement scale, and appropriate sets of relative risks were calculated. The adjustment changed the relative risk pattern very little.

Residential mobility determines pollution exposures and may itself influence the incidence of acute illness, since changes in life situations seem to increase morbidity from diverse causes.[12] Mobility and social status are themselves interrelated in that socioeconomically advantaged upper-middle-class

### Table 4.3.6. RELATIVE SEVERITY SCORES FOR ACUTE RESPIRATORY ILLNESS

| Family segment | Community | Relative severity score[a] Upper tract | Lower tract | Total |
|---|---|---|---|---|
| Fathers | Intermediate | 1.00 (1.63) | 1.00 (2.57) | 1.00 (1.76) |
| | High | 0.92 | 1.47 | 1.21 |
| | Highest | 0.91 | 1.38 | 1.12 |
| Mothers | Intermediate | 1.00 (1.59) | 1.00 (3.23) | 1.00 (1.82) |
| | High | 0.97 | 0.75 | 0.88 |
| | Highest | 0.91 | 0.74 | 0.86 |
| Older siblings | Intermediate | 1.00 (2.13) | 1.00 (3.82) | 1.00 (2.33) |
| | High | 1.08 | 1.66 | 1.31 |
| | Highest | 1.03 | 1.07 | 0.99 |
| Nursery school children | Intermediate | 1.00 (2.26) | 1.00 (4.27) | 1.00 (2.62) |
| | High | 0.87 | 0.77 | 0.93 |
| | Highest | 0.92 | 0.91 | 1.04 |
| Younger siblings | Intermediate | 1.00 (1.99) | 1.00 (3.79) | 1.00 (2.62) |
| | High | 0.89 | 0.77 | 1.12 |
| | Highest | 1.08 | 0.97 | 0.93 |

[a] Base severity score in parentheses.

### Table 4.3.7. RESTRICTED ACTIVITY ACCOMPANYING SEVERITY SCORES FOR ACUTE RESPIRATORY ILLNESS AMONG RESIDENTIALLY STABLE FAMILIES

| Family segment | Severity score | Mean number of restricted activity days at each level of severity: Upper tract | Lower tract | Total |
|---|---|---|---|---|
| Parents | I | 0 | 0 | 0 |
| | II | 2.11 | 3.05 | 2.27 |
| | III | 2.32 | 3.33 | 2.54 |
| | IV | 4.34 | 5.00 | 4.68 |
| Older siblings | I | 0 | 0 | 0 |
| | II | 2.11 | 2.62 | 2.21 |
| | III | 1.95 | 2.20 | 2.00 |
| | IV | 3.53 | 4.24 | 3.67 |
| | V | 5.60 | 3.00[a] | 5.17 |
| Nursery school children and younger siblings | I | 0 | 0 | 0 |
| | II | 2.38 | 3.78 | 2.75 |
| | III | 2.14 | 4.00 | 2.58 |
| | IV | 3.77 | 5.24 | 4.23 |
| | V | 4.73 | 4.50 | 4.69 |

[a] Unusual mean based on one illness.

Table 4.3.8. EXCESS RESTRICTED ACTIVITY NECESSITATED BY MORE FREQUENT OR MORE SEVERE ACUTE RESPIRATORY ILLNESS

| Family segment | Excess restricted activity days per 100 person-weeks of risk | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract | | Lower tract | | Total | |
| | Percent excess | Excess days | Percent excess | Excess days | Percent excess | Excess days |
| Fathers | -13 | 0.47 | 97 | 1.36 | 18 | 0.89 |
| Mothers | -14 | 0.74 | 40 | 1.06 | 23 | 1.80 |
| Older siblings | 2 | 0.13 | 23 | 0.52 | 6 | 0.65 |
| Nursery school children | 4 | 0.68 | 40 | 3.33 | 15 | 4.01 |
| Younger siblings | 20 | 3.71 | 9 | -1.24 | 8 | 2.47 |
| Total for nuclear family of five | 9 | 4.79 | 17 | 5.03 | 12 | 9.82 |

Table 4.3.9. SEVERITY OF ACUTE RESPIRATORY ILLNESS IN RESIDENTIALLY STABLE FAMILIES

| Family segment | Community | Percent of acute respiratory illnesses | | |
|---|---|---|---|---|
| | | Requiring restricted activity | Requiring physician visit | Accompanied by physician-diagnosed otitis media |
| Fathers | Intermediate | 38 | 10 | – |
| | Highest | 49 | 18 | – |
| Mothers | Intermediate | 34 | 7 | – |
| | Highest | 32 | 7 | – |
| Older siblings | Intermediate | 58 | 24 | 1.9 |
| | Highest | 62 | 30 | 1.1 |
| Nursery school children | Intermediate | 60 | 27 | 3.5 |
| | Highest | 56 | 26 | 4.9 |
| Younger siblings | Intermediate | 60 | 25 | 1.5 |
| | Highest | 57 | 26 | 8.3 |

### Table 4.3.10. REPEATED ACUTE RESPIRATORY ILLNESSES AMONG MEMBERS OF RESIDENTIALLY STABLE FAMILIES

| Family segment | Community | Percent of members with repeated acute respiratory illnesses | | |
|---|---|---|---|---|
| | | More than 3 upper tract illnesses | More than 1 lower tract illness | More than 4 total illnesses |
| Fathers | Intermediate | 6 | 5 | 6 |
| | Highest | 8 | 26 | 14 |
| Mothers | Intermediate | 20 | 4 | 15 |
| | Highest | 35 | 6 | 21 |
| Older siblings | Intermediate | 24 | 5 | 17 |
| | Highest | 33 | 13 | 33 |
| Nursery school children and younger siblings | Intermediate | 49 | 21 | 49 |
| | Highest | 56 | 20 | 56 |

families seem more mobile than less advantaged working-class families. Residentially stable families were defined as those who had not changed addres during the preceeding 3 years. Acute respiratory disease attack rates for each family segment were compiled for mobile and residentially stable families, and sets of relative risks were calculated as previously described (Table 4.3.11). The most striking result of the length-of-residence partition was an increase in the magnitude of the intercommunity differences in acute respiratory illness found among the residentially stable families. These excesses, most of which were statistically significant (Table 4.3.12), could be attributed to long-term exposures to air pollution. On the other hand, attack rates among the more mobile population were quite variable, and overall there seemed to be little difference except for a questionable increase in total acute respiratory illness among children exposed to the highest level of ambient air pollution. The analysis suggested that the excess morbidity might either be due to exposures lasting 3 years or exposures of indeterminate length involving ambient levels occurring 1 or 2 years before the study in the most polluted community.

To determine whether educational attainment and mobility were related in some complex fashion that would bias the study, attack rates were compiled for residentially stable families and partitioned into two educational attainment groups on the basis of graduate school attendance (Table 4.3.13). Attack rates were relatively unstable because of small sample sizes, but a clear pattern was apparent: excesses in illness attributable to pollution were greater among the economically advantaged. While this finding might tend to diminish the differences attributed to pollution by the data in Table 4.3.12, the differences were hardly of a magnitude to account for the entire pollution effect. Educational differences exerted a statistically significant influence only in the case of upper tract illness (Table 4.3.12).

Cigarette smoking constitutes an intense self-pollution hazard and contributes to indoor air pollution for members of the family who do not smoke. Since the Intermediate and Highest communities reported similar smoking habits, it was unlikely that this factor could bias the study. Nevertheless, smoking-specific attack rates were calculated for acute respiratory illness, and relative risk models were computed for each family segment (Table 4.3.14). Cigar and pipe smokers were not included in the smoking category. Among mothers, current cigarette smokers seem to experience somewhat more illness than lifetime

### Table 4.3.11. RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS AMONG STABLE AND MOBILE FAMILIES[a]

| Family segment and community | Relative risk of acute respiratory illness[b] | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract | | Lower tract | | Total | |
| | Mobile | Stable | Mobile | Stable | Mobile | Stable |
| Fathers | | | | | | |
| Intermediate | 1.27 | 1.00 (2.59) | 1.10 | 1.00 (0.42) | 1.25 | 1.00 (3.00) |
| Highest | 1.20 | 1.21 | 0.90 | 2.29 | 1.14 | 1.36 |
| Mothers | | | | | | |
| Intermediate | 1.14 | 1.00 (3.96) | 1.53 | 1.00 (0.64) | 1.19 | 1.00 (4.60) |
| Highest | 1.24 | 1.46 | 1.22 | 1.45 | 1.28 | 1.46 |
| Older siblings | | | | | | |
| Intermediate | 1.14 | 1.00 (4.09) | 0.81 | 1.00 (0.63) | 1.09 | 1.00 (4.73) |
| Highest | 1.17 | 1.37 | 0.82 | 1.44 | 1.25 | 1.80 |
| Nursery school children | | | | | | |
| Intermediate | 1.05 | 1.00 (7.57) | 1.25 | 1.00 (1.63) | 1.09 | 1.00 (9.20) |
| Highest | 1.21 | 1.12 | 1.53 | 1.30 | 1.24 | 1.15 |
| Younger siblings | | | | | | |
| Intermediate | 1.16 | 1.00 (7.65) | 0.90 | 1.00 (2.84) | 1.09 | 1.00 (10.49) |
| Highest | 1.65 | 1.27 | 1.00 | 0.93 | 1.43 | 1.18 |

[a] Mobile families had moved within the past 3 years; stable families had not.

[b] Base rate per 100 person-weeks in parentheses.

nonsmokers, but this did not prove true for fathers. Mothers who were exsmokers also appeared to have a somewhat greater relative risk when compared to lifetime nonsmokers. Assessing the effect of smoking in the home on children was more difficult. Attempts to construct dose-response gradients for smoking in the home were partially frustrated by small sample sizes. Families could, however, easily be divided into those with no smoking in the home and those with smokers. The latter families were divided into homes where the mother smoked and an indeterminant category where the mother was a nonsmoker and either the father was a current light smoker or someone else in the home smoked. When the appropriate relative risk models were constructed, there was a definite increase in the risk of acute lower respiratory illness in the youngest children that could be associated with cigarette smoking in the home. This difference was significant at the $p < 0.05$ level, as shown in Table 4.3.12. These children may be in repeated, intimate contact with indoor air pollution caused by side-stream cigarette smoke from cigarettes left burning by their busy mothers. Evaluation of the smoking-specific rates left some confusion as to the impact upon overall acute lower respiratory attack rates. To answer the question, smoking-adjusted acute lower respiratory attack rates were calculated (Table 4.3.15). The adjustment further strengthened the relative risks attributable to pollution in children and in adults.

Table 4.3.12. PROBABILITY SUMMARY FOR EFFECTS OF SELECTED DETERMINANTS ON ACUTE RESPIRATORY DISEASE FREQUENCY AMONG RESIDENTIALLY STABLE FAMILIES

| | | | Significance[a] | | |
|---|---|---|---|---|---|
| Determinant | Study period | Family segment | Upper tract | Lower tract | Total |
| Pollution exposure | Overall | All members | $<0.05$ | $<0.05$ | $<0.005$ |
| | | Parents | $<0.025$ | NS | $<0.01$ |
| | | Children | NS | $<0.05$ | $<0.05$ |
| | Influenza | All members | $<0.10$ | NS | $<0.025$ |
| | Noninfluenzal respiratory | All members | $<0.025$ | NS | $<0.005$ |
| | Summer | All members | NS | $<0.05$ | $<0.10$ |
| Socioeconomic status | Overall | All members | $<0.001$ | NS | NS |
| Personal cigarette smoking | Overall | Parents | NS | $<0.10$ | NS |
| Cigarette smoking in home | Overall | Children | $<0.10$ | $<0.001$ | $<0.05$ |

[a] NS – not significant, $p > 0.10$. All significant associations involve an increase in illness frequency.

## Age-specific Attack Rates

Age-specific acute respiratory attack rates among children from residentially stable families (Figure 4.3.2) can be used to estimate the benefits of recent pollution control or the length of exposure necessary to increase the vulnerability of a child to acute lower respiratory illness.

Children from the Highest exposure community were found to have higher attack rates during the first 3 years of life. This finding suggests that infants living in the Intermediate exposure community benefit substantially from control of sulfur dioxide. There was a decline in attack rates after 3 years of age, and intercommunity differences were quite small or nonexistent. A possible explanation for this observation may be that the child who first enters nursery school or elementary school is already maximally challenged by a succession of infectious agents and that little or no further increase in illness can be attributed to the more subtle environmental insults.

## DISCUSSION

This study corroborates the findings of Douglas and Waller[4] and Lunn et al.[5] Acute respiratory morbidity was significantly lower among families living in neighborhoods where sulfur dioxide levels had been substantially decreased. Furthermore, the most striking improvements involved reductions in total acute respiratory illness among the youngest children and acute lower respiratory illness among adults.

The gradient of pollution exposure developed for this study was based on total suspended particulate concentrations. As a result, some families were included in the Intermediate community although

**Table 4.3.13. EFFECT OF EDUCATIONAL ATTAINMENT UPON RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS IN RESIDENTIALLY STABLE FAMILIES**

| Family segment and community | Relative risk of acute respiratory illness[a] | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract | | Lower tract | | Total | |
| | High socioeconomic status | Low socioeconomic status | High socioeconomic status | Low socioeconomic status | High socioeconomic status | Low socioeconomic status |
| Fathers | | | | | | |
| Intermediate | 1.00 (2.35) | 1.03 | 1.00 (0.43) | 0.91 | 1.00 (2.78) | 1.01 |
| Highest | 1.49 | 0.85 | 2.09 | 1.40 | 1.58 | 0.94 |
| Mothers | | | | | | |
| Intermediate | 1.00 (4.30) | 0.92 | 1.00 (0.43) | 2.16 | 1.00 (4.73) | 1.04 |
| Highest | 1.37 | 0.93 | 2.02 | 1.65 | 1.43 | 1.00 |
| Older siblings | | | | | | |
| Intermediate | 1.00 (5.02) | 0.80 | 1.00 (0.56) | 1.30 | 1.00 (5.58) | 0.85 |
| Highest | 1.27 | 0.64 | 3.57 | 0.55 | 1.58 | 0.63 |
| Nursery school children | | | | | | |
| Intermediate | 1.00 (8.76) | 0.80 | 1.00 (1.73) | 0.80 | 1.00 (10.50) | 0.80 |
| Highest | 1.03 | 0.65 | 1.26 | 1.39 | 1.06 | 0.77 |
| Younger siblings | | | | | | |
| Intermediate | 1.00 (6.18) | 1.15 | 1.00 (3.09) | 0.79 | 1.00 (9.27) | 1.03 |
| Highest | 1.89 | 0.97 | 0.65 | 1.17 | 1.48 | 1.04 |

[a] Base rate for 100 person-weeks of risk in parentheses.

they were exposed to sulfur dioxide levels higher than those of some families in the High and Highest communities. However, a separate analysis of those families with higher sulfur dioxide exposures showed that each population segment, with the exception of the youngest children, had respiratory disease rates that were higher than the mean rate for the Intermediate community. Thus, inclusion of these families in the Intermediate community would tend to obscure differences in disease rates between the communities that could be attributed to pollutant levels.

No lessening of illness severity could be documented, except for a decrease in otitis media among the youngest children living in the Intermediate community. Lessened illness frequency in the community with improved air quality combined with unchanged severity resulted in substantial reductions in the restricted activity burden associated with acute respiratory illness. Another important finding was that reductions in ambient air pollution might reduce the vulnerability of families to an epidemic outbreak of influenza.

Residential mobility, socioeconomic status, and cigarette smoking in the home were shown to be determinants of acute lower respiratory disease in children, but none of these factors accounted for the excesses attributed to ambient air pollution. Another potential indoor air pollution problem, nitrogen dioxide emissions from cooking gas, could hardly bias the study since there were no significant intercommunity differences in the use of domestic gas for cooking or space heating.

It would be a most serious error to assert that the present study demonstrated the full benefits of air

## Table 4.3.14. EFFECT OF CIGARETTE SMOKING UPON RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS IN RESIDENTIALLY STABLE FAMILIES

| Family segment | Community | Current personal or family cigarette smoking status | Relative risk of acute respiratory illness[a] | | |
|---|---|---|---|---|---|
| | | | Upper tract | Lower tract | Total |
| Fathers | Intermediate | Lifetime nonsmoker | 1.00 (2.71) | 1.00 (0.27) | 1.00 (2.98) |
| | Highest | Lifetime nonsmoker | 1.28 | 4.44 | 1.57 |
| | Intermediate | Exsmoker | 0.89 | 1.65 | 0.97 |
| | Highest | Exsmoker | 0.89 | 1.59 | 0.94 |
| | Intermediate | Current cigarette smoker | 0.73 | 2.14 | 0.87 |
| | Highest | Current cigarette smoker | 0.96 | 1.48 | 1.00 |
| Mothers | Intermediate | Lifetime nonsmoker | 1.00 (3.78) | 1.00 (0.52) | 1.00 (4.30) |
| | Highest | Lifetime nonsmoker | 1.46 | 1.38 | 1.45 |
| | Intermediate | Exsmoker | 2.51 | 1.92 | 2.44 |
| | Highest | Exsmoker | 2.64 | 21.15[b] | 2.32 |
| | Intermediate | Current cigarette smoker | 1.09 | 1.98 | 1.20 |
| | Highest | Current cigarette smoker | 1.06 | 2.10 | 1.18 |
| Older siblings | Intermediate | No current smoking | 1.00 (4.84) | 1.00 (0.32) | 1.00 (5.16) |
| | Highest | No current smoking | 0.83 | 5.18 | 1.14 |
| | Intermediate | Indeterminant | 0.57 | 1.78 | 0.65 |
| | Highest | Indeterminant | 2.13 | 4.47 | 2.27 |
| | Intermediate | Mother smokes | 0.99 | 3.06 | 1.12 |
| | Highest | Mother smokes | 0.41 | 0.00 | 0.39 |
| Nursery school children | Intermediate | No current smoking | 1.00 (8.56) | 1.00 (1.72) | 1.00 (10.28) |
| | Highest | No current smoking | 0.80 | 1.78 | 0.96 |
| | Intermediate | Indeterminant | 0.86 | 1.03 | 0.89 |
| | Highest | Indeterminant | 1.14 | 1.16 | 1.14 |
| | Intermediate | Mother smokes | 0.84 | 0.67 | 0.81 |
| | Highest | Mother smokes | 0.83 | 0.77 | 0.81 |
| Younger siblings | Intermediate | No current smoking | 1.00 (7.82) | 1.00 (2.18) | 1.00 (10.00) |
| | Highest | No current smoking | 0.93 | 1.15 | 0.98 |
| | Intermediate | Indeterminant | 0.55 | 0.77 | 0.60 |
| | Highest | Indeterminant | 2.05 | 0.46 | 1.70 |
| | Intermediate | Mother smokes | 0.90 | 1.68 | 1.07 |
| | Highest | Mother smokes | 1.41 | 2.29 | 1.60 |

[a] Base rate per 100 person-weeks of risk is in parentheses.

[b] Unstable risk based on a very small sample.

## 4.3.15. SMOKING-ADJUSTED RELATIVE RISK OF ACUTE RESPIRATORY DISEASE IN RESIDENTIALLY STABLE FAMILIES

| Family segment | Community | Relative risk of acute respiratory illness[a] | | |
|---|---|---|---|---|
| | | Upper tract | Lower tract | Total |
| Fathers | Intermediate | 1.00 (2.39) | 1.00 (0.41) | 1.00 (2.80) |
| | Highest | 1.25 | 1.90 | 1.33 |
| Mothers | Intermediate | 1.00 (4.06) | 1.00 (0.70) | 1.00 (4.76) |
| | Highest | 1.27 | 1.64 | 1.25 |
| Older siblings | Intermediate | 1.00 (4.40) | 1.00 (2.64) | 1.00 (7.04) |
| | Highest | 1.01 | 1.46 | 1.18 |
| Nursery school students | Intermediate | 1.00 (7.82) | 1.00 (1.53) | 1.00 (0.35) |
| | Highest | 0.99 | 1.16 | 1.02 |
| Younger siblings | Intermediate | 1.00 (6.87) | 1.00 (2.54) | 1.00 (9.41) |
| | Highest | 1.48 | 1.09 | 1.37 |

[a] Base rate per 100 person-weeks at risk is in parentheses.

pollution control on acute respiratory morbidity. The limited nature of air monitoring data precluded quantifying possible short-term peak exposure effects. The age-specific attack rate pattern presented is consistent with the emergence of increased susceptibility to acute respiratory illness after less than 3 years of exposures to elevated pollutant levels. Perhaps less than 1 year of exposure is required. Such exposures cannot be fully evaluated without a comprehensive monitoring of short-term pollutant levels. Another problem relates to the confidence with which a pollutant or pollutants might be incriminated as causal agents for the observed impairments. The evidence indicates the Intermediate community most likely benefitted from reductions in sulfur dioxide and perhaps suspended sulfates. Suspended particulate reduction, although more modest, could well have played a role in the observed improvements.

After weighing the strengths and limitations of the present study, we concluded that there was strong evidence that reducing sulfur dioxide levels from an annual average somewhat above the relevant National Primary Air Quality Standard to a level below the standard (107 to 109 $\mu g/m^3$ compared with 51 to 57 $\mu g/m^3$) would reduce acute respiratory disease morbidity. Reductions in total suspended particulate and/or suspended sulfate levels could well result in additional reductions in acute respiratory illness, but the reductions in illness associated with reductions in sulfur dioxide are felt to be distinct from those that might result from reductions in other pollutants.

## SUMMARY

A prospective survey of over 2500 members of volunteer families indicated that those individuals living more than 3 years in high pollution areas (sulfur dioxide, 107 to 250 $\mu g/m^3$; total suspended particulates. 137 to 165 $\mu g/m^3$) had significantly increased rates of acute respiratory illness, restricted activity, and otitis media when compared to individuals living in less polluted areas (sulfur dioxide, 109 to 130 $\mu g/m^3$; total suspended particulates, 121 to 123 $\mu g/m^3$). It is also possible that more recent lower air pollution levels contributed to increased respiratory illness. During the study, sulfur dioxide levels in the lower pollution areas were around 57 $\mu g/m^3$ as compared with 51 to 106 $\mu g/m^3$ in the higher pollution areas; for total suspended particu-

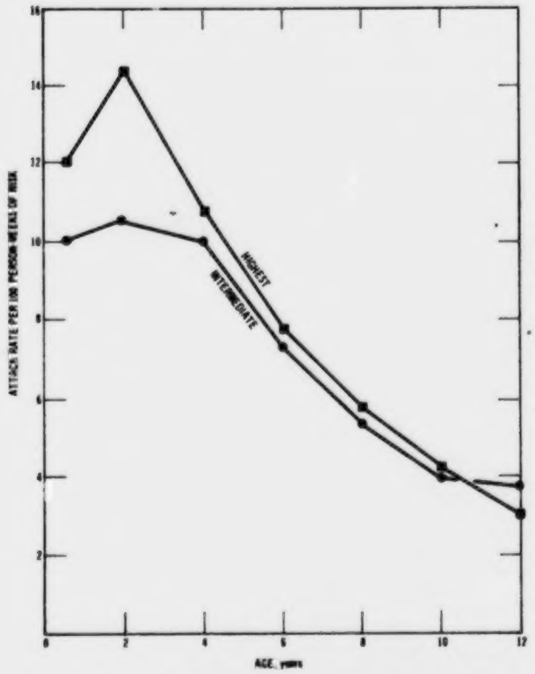

Figure 4.3.2. Age-specific attack rates for acute respiratory illness among children from residentially stable families.

lates, the levels are around 111 $\mu g/m^3$ as compared with 126 to 151 $\mu g/m^3$.

Children and adults living in neighborhoods with cleaner air were measurably less susceptible to etiologically undefined illness and to epidemic influenza as well. Families with higher socioeconomic status generally reported more illness. Residential mobility proved a significant determinant of illness, and cigarette smoking by mothers seemed related to increased susceptibility among the youngest preschool children. None of these intervening variables could account for the effects attributed to ambient air pollutants. It was concluded that improvements in air quality can significantly reduce the incidence of respiratory illnesses.

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# CHAPTER 5

# NEW YORK STUDIES

# 5.1 HUMAN EXPOSURE TO AIR POLLUTION IN SELECTED NEW YORK METROPOLITAN COMMUNITIES, 1944-1971

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## INTRODUCTION

The New York metropolitan area, with over ten million people exposed to a complex mixture of air pollutants, provides a unique setting for studying the influence of pollutants on human health. Although the patterns of pollution in a metropolitan area obviously are much more complex than those of less highly urbanized situations, the size of potential study groups and the size of the population possibly experiencing adverse effects make it imperative that the studies be undertaken. In the metropolitan area, measurement of major pollutants–sulfur dioxide ($SO_2$), total suspended particulates (TSP), suspended nitrates (SN), suspended sulfates (SS), and dustfall–provides useful indices of total pollution that can be used as a basis for comparison with other areas. The major metropolitan areas are also the locations in which improvements in air quality are most likely to be effected at an early date. It follows then that these areas will be the first in which data related to the benefits of control can be obtained. For these reasons, the Community Health and Environmental Surveillance (CHESS) program of the Environmental Protection Agency (EPA) was initiated in the New York area in April 1970.

### Community Description

Within the New York City metropolitan area, three communities were selected on the basis of several years of prior data to comprise a gradient of exposure to sulfur oxides and total suspended particulates. These are the Westchester sector of the Bronx, a community expected to have high exposures to both sulfur oxides and total suspended particulate; the Howard Beach Section of the Queens, an intermediate exposure community; and Riverhead, Long Island, a low exposure community.

The New York CHESS area is characterized by a flat terrain with elevations of 100 feet or less above sea level. Bronx, Queens, and Riverhead, are all located near rivers or bays (Figure 5.1.1). The climate of the area can best be defined as modified continental. Weather situations that make up the climate pattern move from a generally westerly direction. The maritime influence is shown by the uniform occurrence of precipitation throughout the year in contrast to the summertime concentration of rainfall that is typical of an inland regime. The maritime influence also moderates temperatures in a manner that produces differences between "in-city" and suburban locations that are often of considerable magnitude.

Other factors influencing the climate of the area are coastal storms that produce strong winds and heavy precipitation and low-pressure systems, also associated with unsettled weather, that frequently pass through the area. The prevailing westerly winds experienced are illustrated by wind roses from John F. Kennedy and LaGuardia airports (Figure 5.1.2 and 5.1.3).

Annual mean temperature in New York City is somewhat higher than that of most places at the same latitude in the United States, except Pacific coastal localities. In summer, New York City may be hot, but extended heat waves are rare. Autumn is milder than spring.

### Pollution Sources

In the metropolitan New York area, numerous and varied pollution sources produce a complex mixture of pollutants, the composition of which has not been defined completely. The measurement of major pollutants is acknowledged to be one means of indexing this total pollution.

Sulfur dioxide and suspended particulates are produced primarily by the combustion of fossil fules and thus by the industrial and domestic units that burn these fuels. Electric power plants have been shown to be the major producers, but

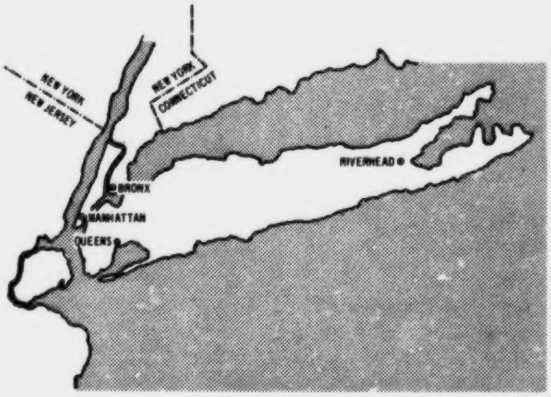

Figure 5.1.1. New York CHESS communities.

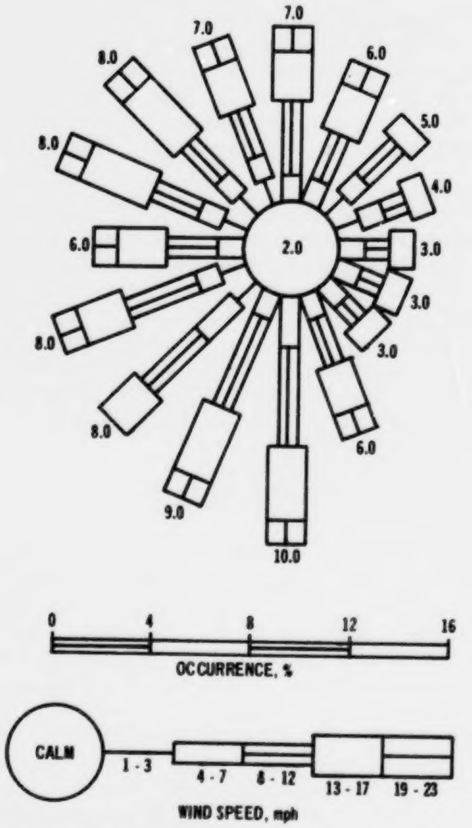

Figure 5.1.2. Surface climatologic wind rose for John F. Kennedy Airport, New York. (Percentage of time wind is in direction shown.)

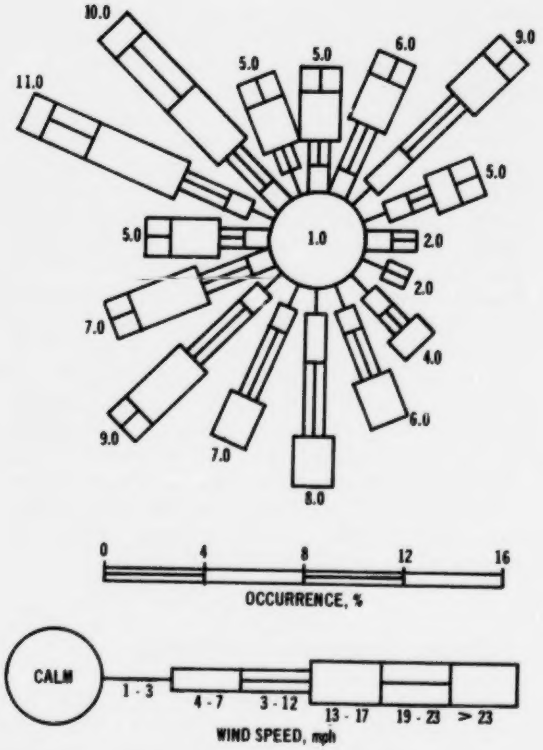

Figure 5.1.3. Surface climatologic wind rose for La Guardia Airport, New York. (Percentage of time wind is in direction shown.)

a variety of other industrial facilities contribute to the total sulfur dioxide emissions. Many of these sources, as well as incinerators and automobiles, contribute significantly to total particulate emissions.

## Monitoring Human Exposure to Air Pollutants

The CHESS strategy employed for monitoring human exposure to environmental pollutants has been described in other CHESS reports.[1,2] The New York populations under surveillance were located within 1.5 miles of the CHESS air monitoring sites.

## Location and Description of Monitoring Sites

All New York monitoring sites were located on tops of buildings 30 to 45 feet above ground level. CHESS stations in New York City were located on the same sites as those of the New York City Department of Air Resources (NYC-DAR). Since the NYC-DAR was also measuring current air quality and had several years of past data available, the analyses in this monograph utilized information from both sources to the fullest extent possible.

Two aspects of the location of the monitoring sites must be considered in relating the data obtained to the exposure of the populations being studied. First, the air monitoring station in the Bronx community was situated on top of a three-story court house in the center of a busy commercial area, while those in the Queens and Riverhead communities were located on two-story buildings surrounded by residential dwellings. Thus, at the Bronx station, proximity to heavy traffic would tend to increase measured pollutant levels, while greater height above ground would tend to decrease these values. The stations in the Queens and Riverhead communities probably more accurately reflected the ambient exposure experienced by nearby residents. Secondly, the Queens community lies about 1 mile west of the John F. Kennedy International Airport; odors and noise from the airport have been the object of many complaints by the residents of that community. Because of budget limitations, hydrocarbons, one of the important classes of pollutants emitted by aircraft engines, were not monitored at any of the CHESS stations.

## Pollutant Measurement Methods

Similar techniques were used by CHESS and NYC-DAR for the measurement of total suspended particulates[3] and particulate dustfall.[4] Total suspended particulate values recorded by NYC-DAR were consistently about 20 percent higher than values recorded by CHESS. To measure concentrations of sulfur dioxide, CHESS used a modification of the West-Gaeke method[5] and the NYC-DAR used a hydrogen peroxide technique.[6] NYC-DAR estimated that 15 percent higher concentrations of sulfur dioxide should be expected with the hydrogen peroxide technique.[7] A detailed comparison of NYC-DAR and CHESS sulfur dioxide measurements is presented later in this paper. The same techniques for determining coefficient of haze (COH) were used by CHESS and NYC-DAR.[8] NYC-DAR also made determinations of total suspended particulate by utilizing a dust count instrument that operated by the principal of light scattering.[9]

Nitrogen dioxide concentrations were measured by CHESS and NYC-DAR using a modification of the Jacobs-Hochheiser method, which at the time of data collection was the procedure accepted by the Federal government. The technique has recently come under much criticism. It is widely held that the Jocobs-Hochheiser method does not give accurate results because of several factors, among which are a fluctuating absorption efficiency and the probability of interference from other environmental pollutants. Nitrogen dioxide data reported in this paper, therefore, should only be used in light of the recent findings concerning the accuracy of the measurement method that was employed.

To estimate the fraction of the total suspended particulate that is respirable, a separate set of measurements was made by CHESS using an Atomic Energy Commission cyclone as a prefilter. This device collects all particles larger than 10 μm, filters out 50 percent of the 3.5-μm particles, and has no effect on 2-μm or smaller particles. The respirable particulates penetrating the cyclone are collected on the backup filter.

## Precision of Measurements

The precision of daily CHESS sulfur dioxide collection and analytical techniques is estimated to be ±27 percent for the arithmetic mean based on duplicate measurements made over a period of 19 months in Birmingham, Alabama.[2] Comparison of monthly CHESS and NYC-DAR sulfur dioxide data over a period of 13 months resulted in a correlation coefficient of 0.57 and a mean relative error of ±25 percent based on monthly arithmetic means (Figure 5.1.4). Part of the relative error is due to differences in measurement method; as mentioned earlier, the hydrogen peroxide method used by NYC-DAR has been estimated to give results about 15 percent higher than actual values.

Precision of CHESS total suspended particulate measurements is estimated to be ±6 percent for the arithmetic mean based on duplicate measurements made over a period of 28 months in Birmingham, Alabama, Charlotte, North Carolina, and Greensboro, North Carolina.[2] A comparison of CHESS and NYC-DAR total suspended particulate data over a period of 1 year gave a correlation coefficient of 0.80 and a mean error ±10 percent (Figure 5.1.5). Recent evidence indicates that CHESS measurements may be approximately 20 percent low due to loss of material from the filter during shipment.

Precision of CHESS total dustfall measurements is estimated to be ±25 percent for the arithmetic mean based on duplicate measurements made over a period of approximately 2-1/2 years in Birmingham, Charlotte, and Greensboro.[2] It is believed that the precision of the total dustfall

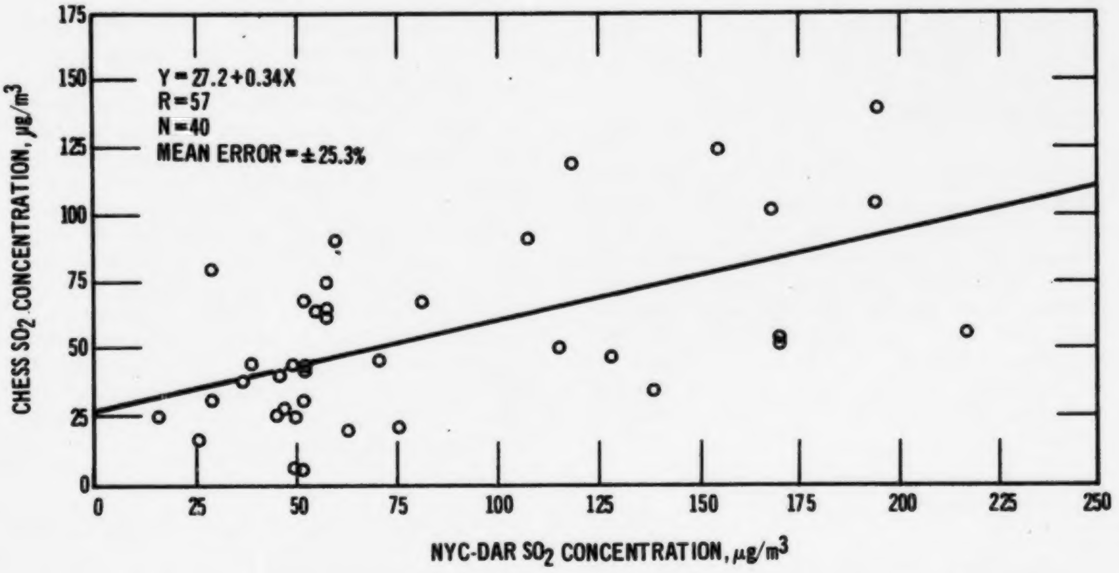

Figure 5.1.4. Comparison of monthly average CHESS and New York City Department of Air Resources sulfur dioxide data for Queens and Bronx.

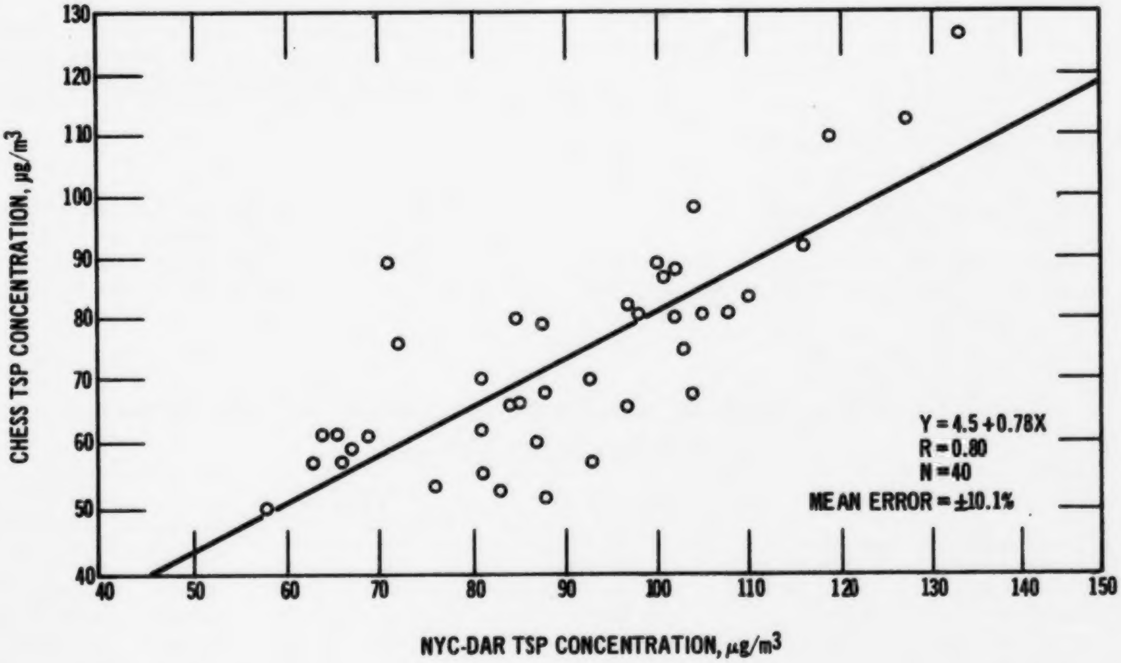

Figure 5.1.5. Comparison of monthly average CHESS and New York City Department of Air Resources total suspended particulate data for Queens and Bronx.

measurement obtained by NYC-DAR is comparable.

### Sampling Frequency

Both CHESS and NYC-DAR data for sulfur dioxide, nitrogen dioxide, and total suspended particulates are based on 24-hour integrated samples. The CHESS respirable suspended particulates measurements are also based on 24-hour samples. Both CHESS and NYC-DAR data for COH are based on 2-hour samples. Dustfall sampling periods are 1 month. A summary of the methods, sampling frequencies, and sampling times for both CHESS and NYC-DAR is given in Table 5.1.1.

### Quality Control

Because data outputs of an air sampling program are subject to many sources of error, an effective quality control program is an essential element of an environmental surveillance system. Effective real-time controls are essential to minimized field errors, systematic drift, chemical laboratory errors, data transfer errors, computer punch card errors, analysis errors, etc. A systematic approach has been used to provide a quality control system for CHESS.[2]

## RESULTS AND DISCUSSION

### Daily Frequency Distribution

Daily frequency distributions for the pollutants measured in the New York communities are given in the Appendix, Tables 5.1.A.1 to 5.1.A.6. Frequency distributions of daily sulfur dioxide concentrations in the three New York communities are illustrated in Figure 5.1.6. In general, the data show a log-normal frequency distribution; however, data for Queens deviate from the straight line for cumulative frequencies greater than 85 percent. Bronx concentrations exceed those in Queens about 15 percent of the time. As a point of reference, the National Annual Primary Ambient Air Quality Standard of 80 $\mu g/m^3$ for sulfur dioxide was exceeded on 5 percent of the days in Riverhead, 20 percent of the days in the Bronx, and 25 percent of the days in Queens. Daily mean values never exceeded 365 $\mu g/m^3$ (the National Daily Primary Ambient Air Quality Standard).

Frequency distributions of daily total suspended particulate concentrations for the New York communities are illustrated in Figure 5.1.7. The data are log-normally distributed, the slopes of the lines for the three areas are approximately the same, and the relative positions of the lines indicate a significant concentration gradient among the three communities. The 24-hour mean total suspended particulate concentrations did not exceed the National Daily Primary Air Quality Standard (260 $\mu g/m^3$) in any of the communities. However, the geometric annual mean for total suspended particulate in the Bronx is slightly above the National Annual Primary Air Quality Standard.

Daily frequency distributions for suspended sulfates in the New York communities (Figure 5.1.8) show that Riverhead generally had lower concentrations and that levels in Bronx and

**Table 5.1.1. METHODS, SAMPLING FREQUENCIES, AND SAMPLING TIMES USED TO MEASURE POLLUTANTS IN NEW YORK AREA**

| Pollutant | Method | | Sampling frequency, samples/yr | | Sampling time | |
|---|---|---|---|---|---|---|
| | NYC-DAR | CHESS | NYC-DAR | CHESS | NYC-DAR | CHESS |
| Sulfur dioxide | Hydrogen peroxide | West-Gaeke | 365 | 365 | 24 hours | 24 hours |
| Nitrogen dioxide | Jacobs-Hochheiser | Jacobs-Hochheiser | 365 | 365 | 24 hours | 24 hours |
| Dustfall | Jar | Bucket | 12 | 12 | Monthly | Monthly |
| Total particulate | High-volume samplers | High-volume samplers | 365 | 365 | 24 hours | 24 hours |
| Smoke shade | AISI | AISI | 4380 | 4380 | 2 hours | 2 hours |
| Dust count | Light scattering | -- | Grab samples | -- | -- | -- |
| Respirable suspended particulate | -- | AEC cyclone | -- | 365 | -- | 24 hours |

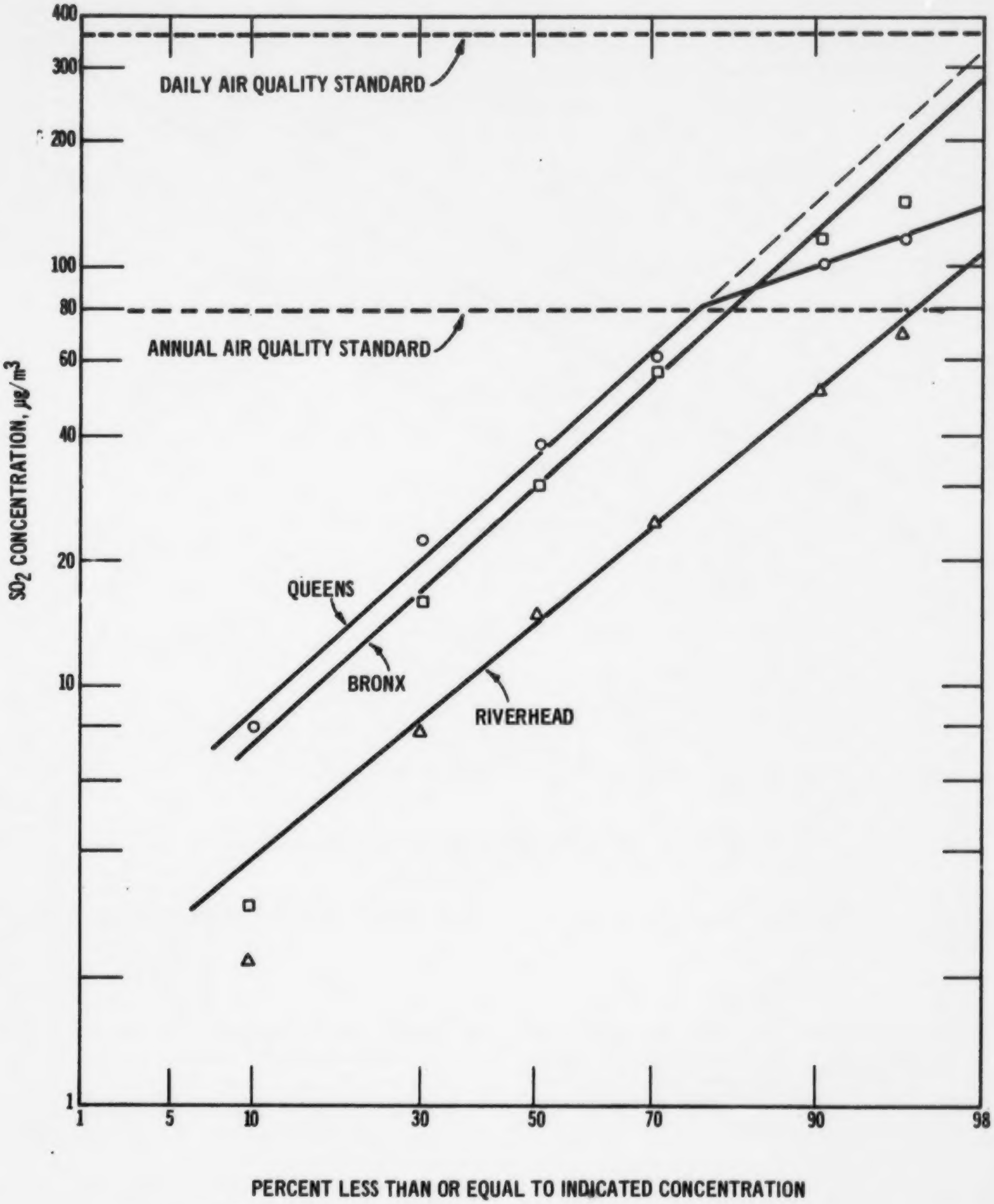

Figure 5.1.6. Cumulative frequency of sulfur dioxide concentrations, New York CHESS, 1971.

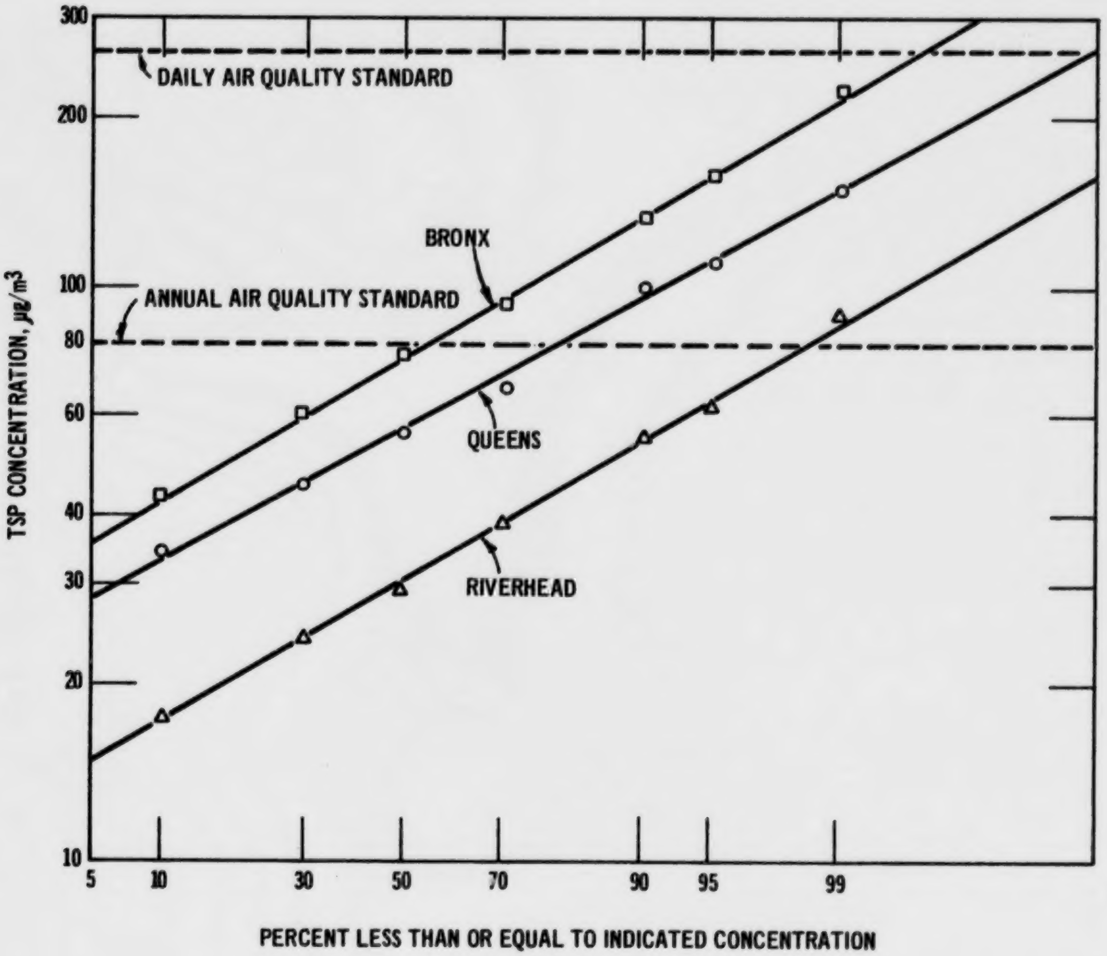

Figure 5.1.7. Cumulative frequency of total suspended particulate concentrations, New York CHESS, 1971.

Queens were similar. Frequency distributions for the nitrate fraction of total suspended particulates, for respirable particulates, and for nitrogen dioxide are not plotted but are given in Tables 5.1.A.4, 5.1.A.5, and 5.1.A.6.

## Seasonal Trends

Quarterly arithmetic averages for sulfur dioxide, nitrogen dioxide, total suspended particulates, suspended sulfates, and suspended nitrates are presented for the three New York CHESS communities in Table 5.1.A.7. These data, from the fourth quarter of 1970 through the first quarter of 1972, represent seasonal averages.

Monthly arithmetic mean sulfur dioxide concentrations for the three New York communities over a 19-month period (Figure 5.1.9) demonstrate an annual cyclic pattern, with peak concentrations occurring in January and minimum values during the summer months. During most of the year, sulfur dioxide levels were highest in Bronx and lowest in Riverhead. Although it is not applicable to monthly means, the National Annual Primary Air Quality Standard is shown in Figure 5.1.9 as a basis for comparison. Six of the 19 monthly mean levels of sulfur dioxide for the Bronx exceeded the Annual Standard, as did 2 of the 19 values for Queens.

Monthly arithmetic mean values for total suspended particulates over a 19-month period do not exhibit an annual cyclic pattern (Figure 5.1.10). The data show a significant gradient, with the highest concentrations in the Bronx and the lowest in Riverhead. Fifteen of 19 mean monthly values from the Bronx exceed the National Annual Primary Ambient Air Quality Standard, as did 2 of the 19 mean values from Queens.

Monthly arithmetic mean values for suspended sulfates over a period of 18 months for New York indicated no seasonal trends (Figure 5.1.11). In general, sulfate levels conform to the gradient observed for total suspended particulate and sulfur dioxide; however, the gradient between the Bronx and Queens is not as well defined. Monthly arithmetic mean values for suspended nitrates in the New York communities are shown in Figure 5.1.12. The gradient is similar to that for suspended sulfates.

Monthly arithmetic mean nitrogen dioxide concentrations exhibited a reasonably consistent

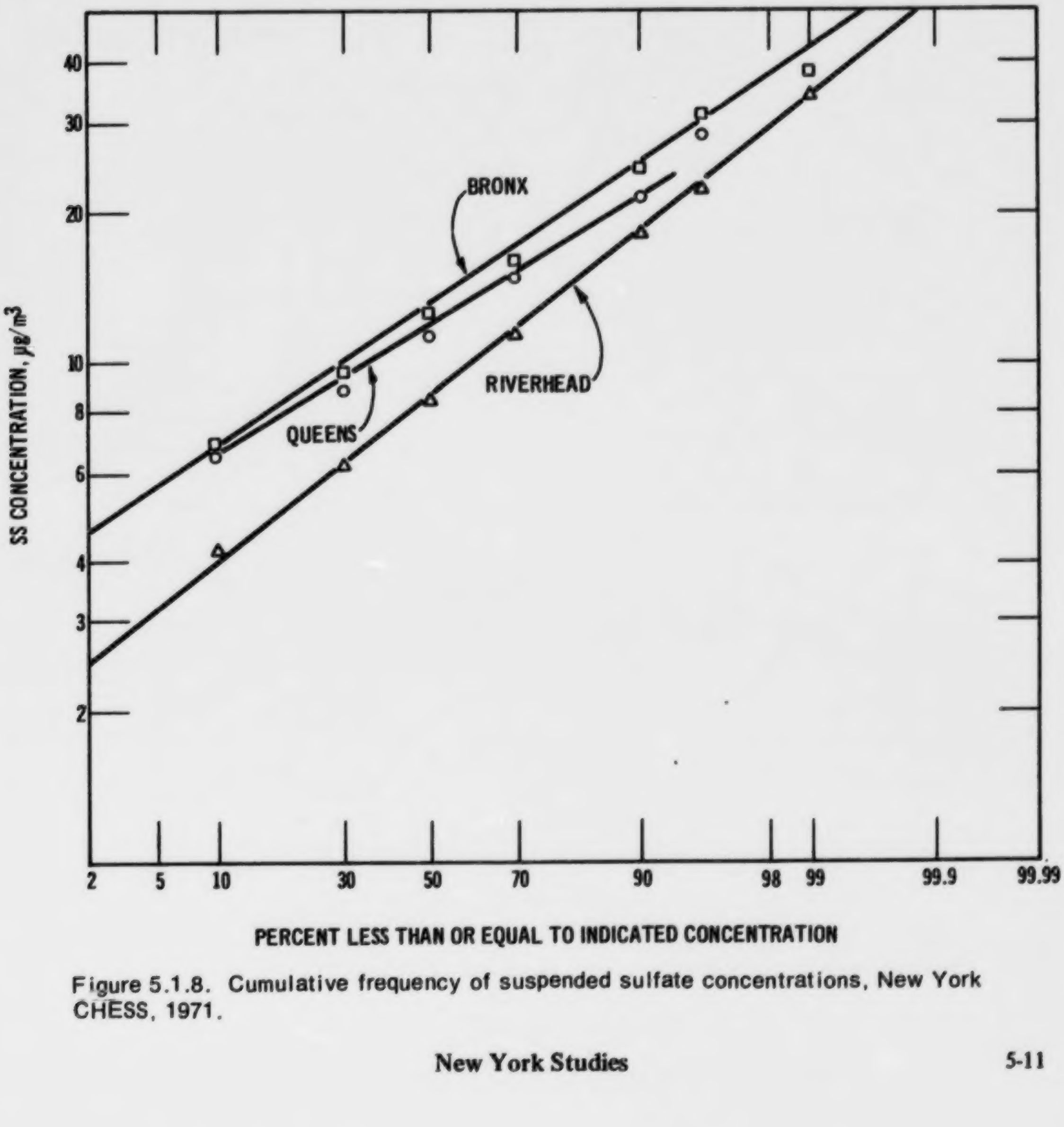

Figure 5.1.8. Cumulative frequency of suspended sulfate concentrations, New York CHESS, 1971.

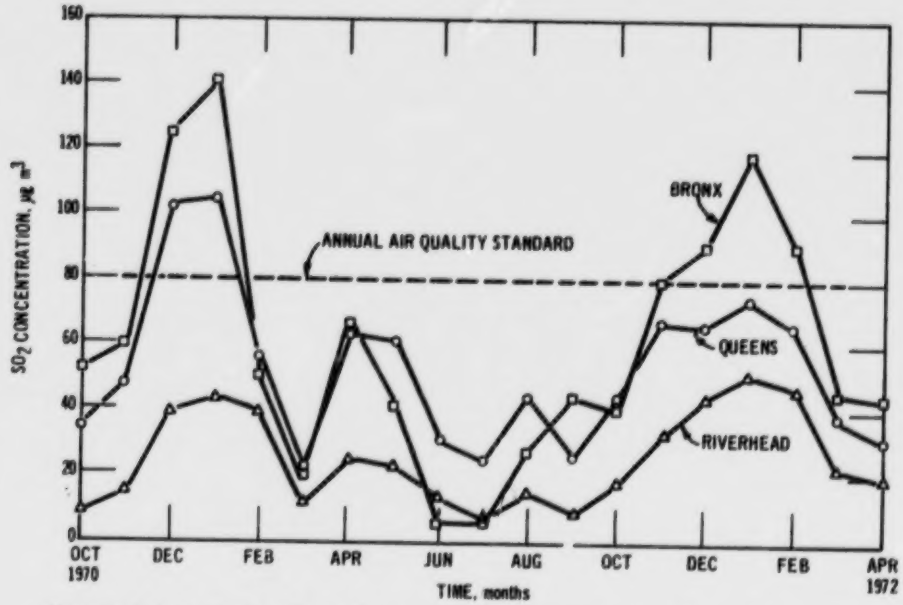

Figure 5.1.9. Sulfur dioxide concentration versus time in months, New York CHESS.

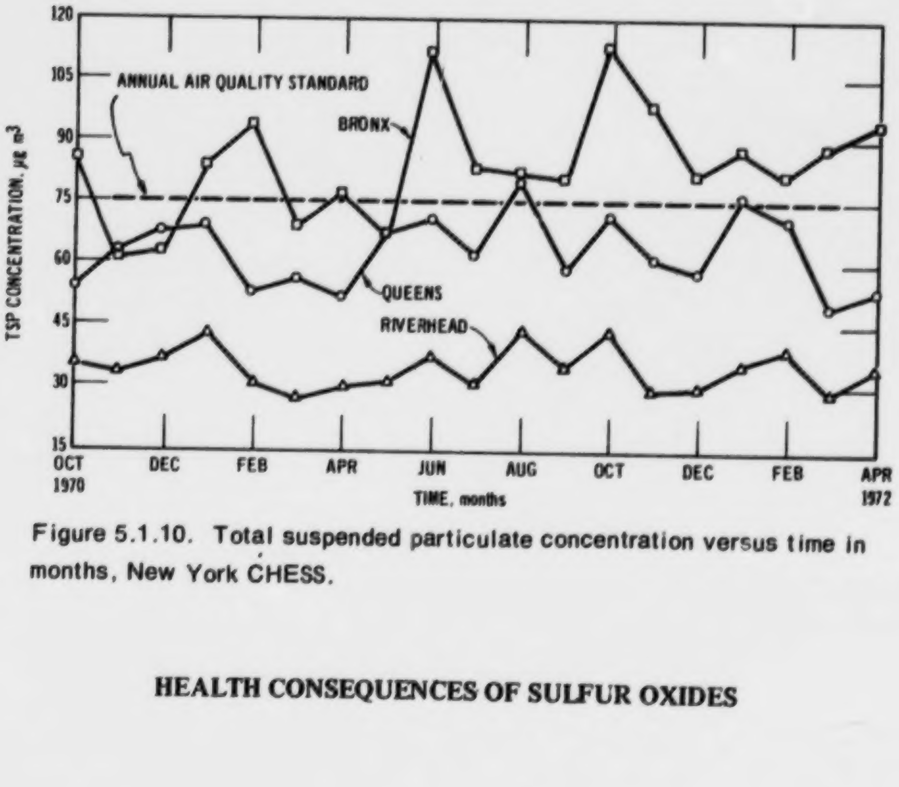

Figure 5.1.10. Total suspended particulate concentration versus time in months, New York CHESS.

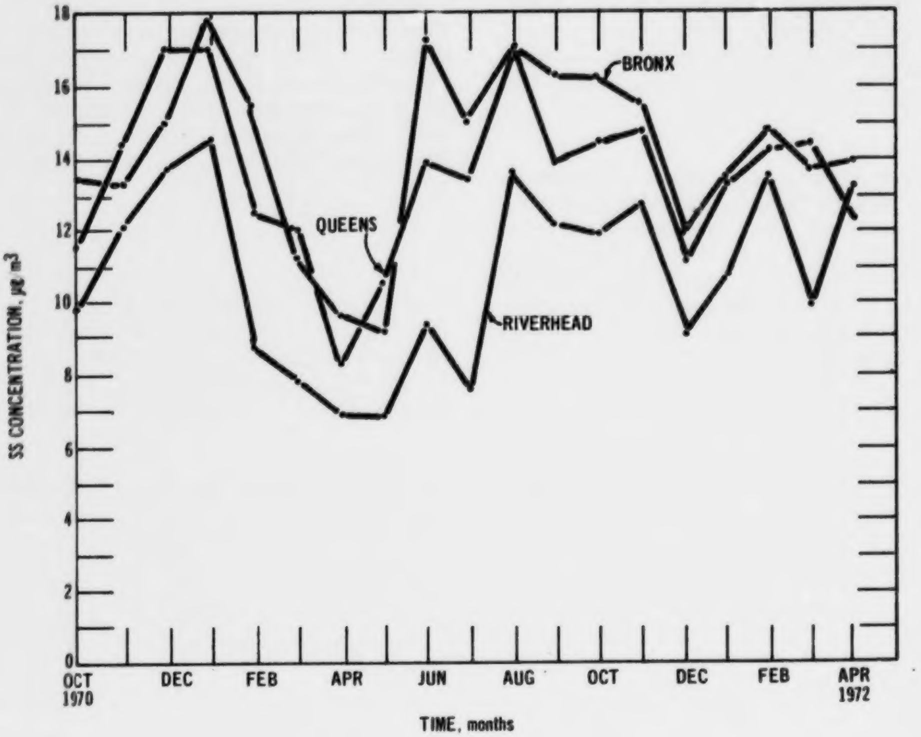

Figure 5.1.11. Suspended sulfate concentration versus time in months, New York CHESS.

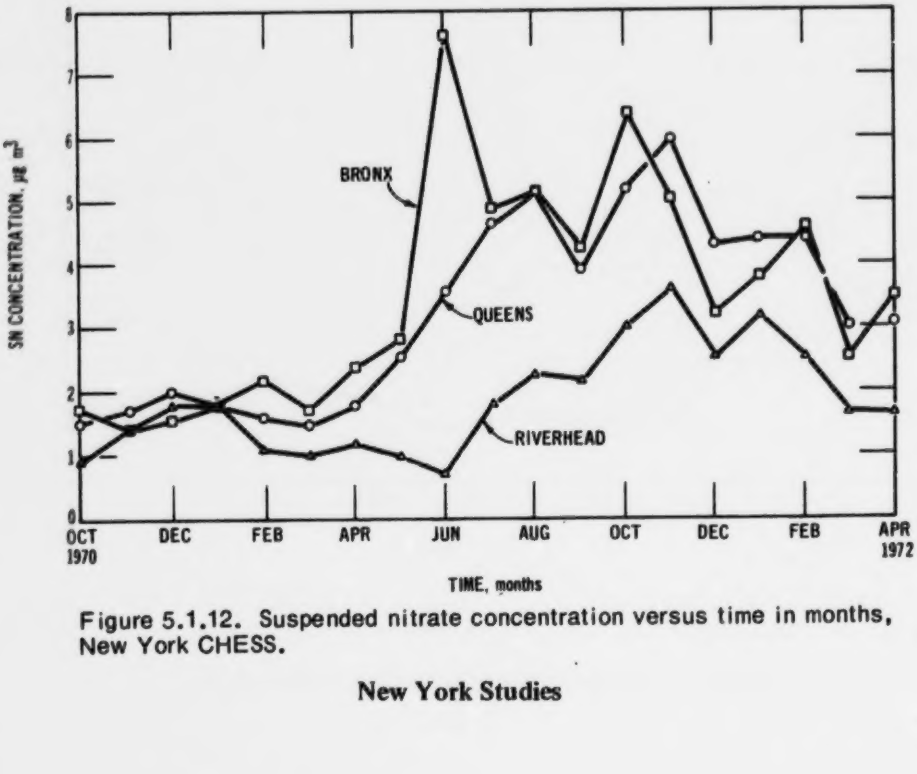

Figure 5.1.12. Suspended nitrate concentration versus time in months, New York CHESS.

gradient for the New York communities (Figure 5.1.13). Since October 1970, none of the monthly mean values have exceeded the National Annual Primary Ambient Air Quality Standard of 100 $\mu g/m^3$.

Monthly arithmetic mean values for respirable suspended particulate (i.e., that portion of total suspended particulates with maximum diameters of 10 $\mu m$) for Bronx, Queens, and Riverhead over a 9-month period (Figure 5.1.14) demonstrated a gradient similar to that of total suspended particulate. These communities are characterized by typical respirable particulate concentrations of about 40 $\mu g/m^3$. The data are insufficient to provide indications of annual cyclic patterns.

Monthly arithmetic mean COH values for the New York communities over a 6-month period (Figure 5.1.15), like the pollutants previously discussed, demonstrate exposure levels that decrease in order from Bronx, to Queens, to Riverhead. The respective average COH values for these communities were 1.25, 0.65, and 0.27.

During most of the year, monthly total dustfall (Figure 5.1.16) also demonstrates a gradient of exposure that decreases in order from Bronx, to Queens, to Riverhead. Cadmium in monthly dustfall samples is much more inconsistent in

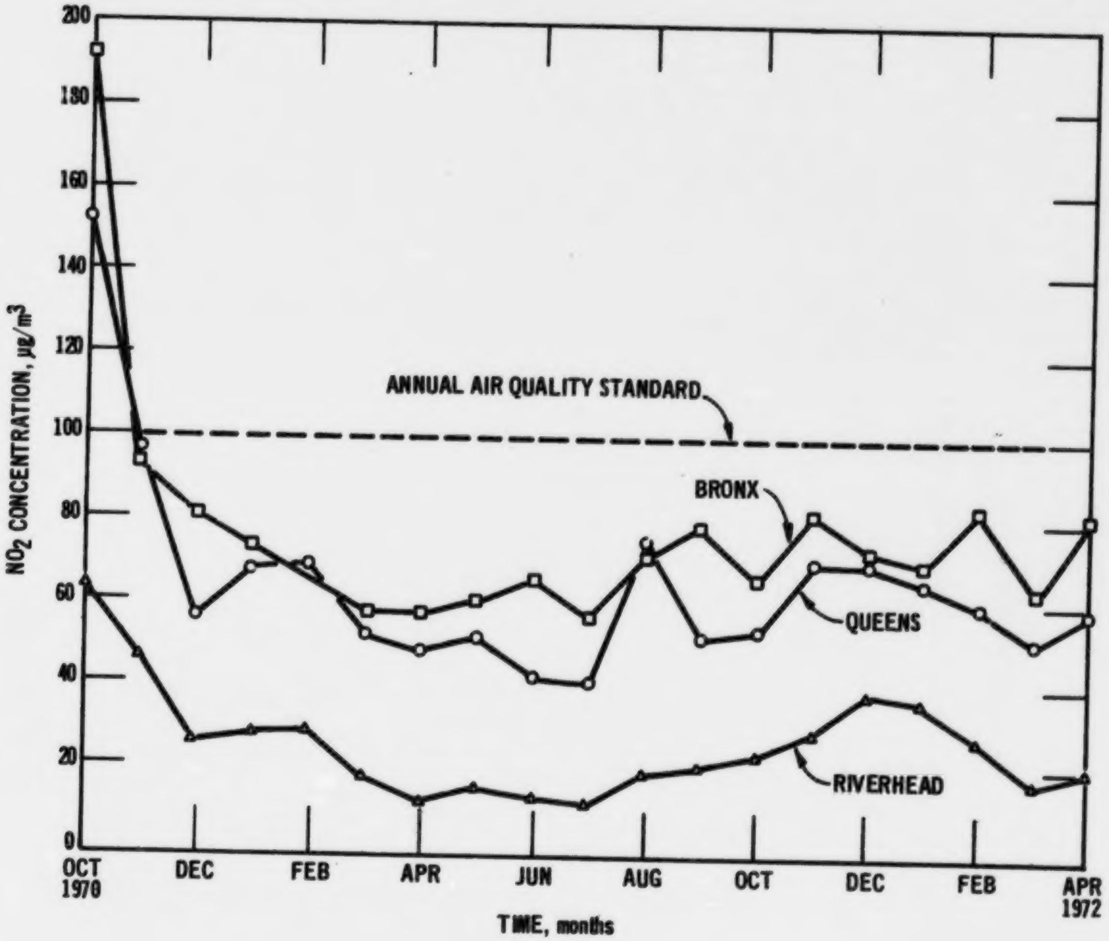

Figure 5.1.13. Nitrogen dioxide concentration versus time in months, New York CHESS.

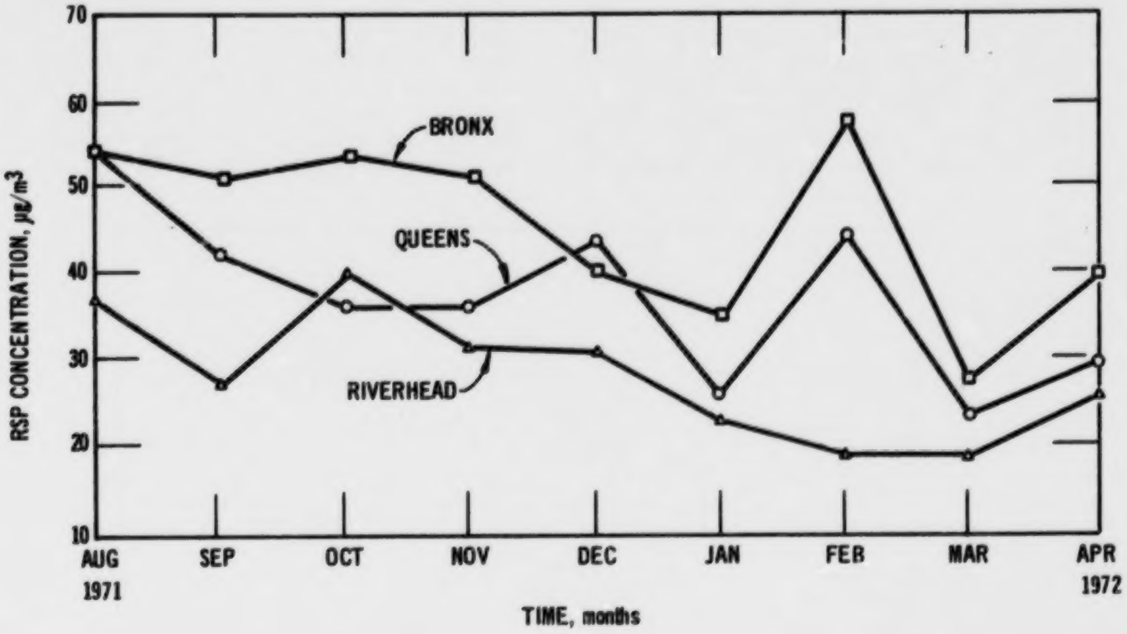

Figure 5.1.14. Respirable suspended particulate concentration versus time in months, New York CHESS.

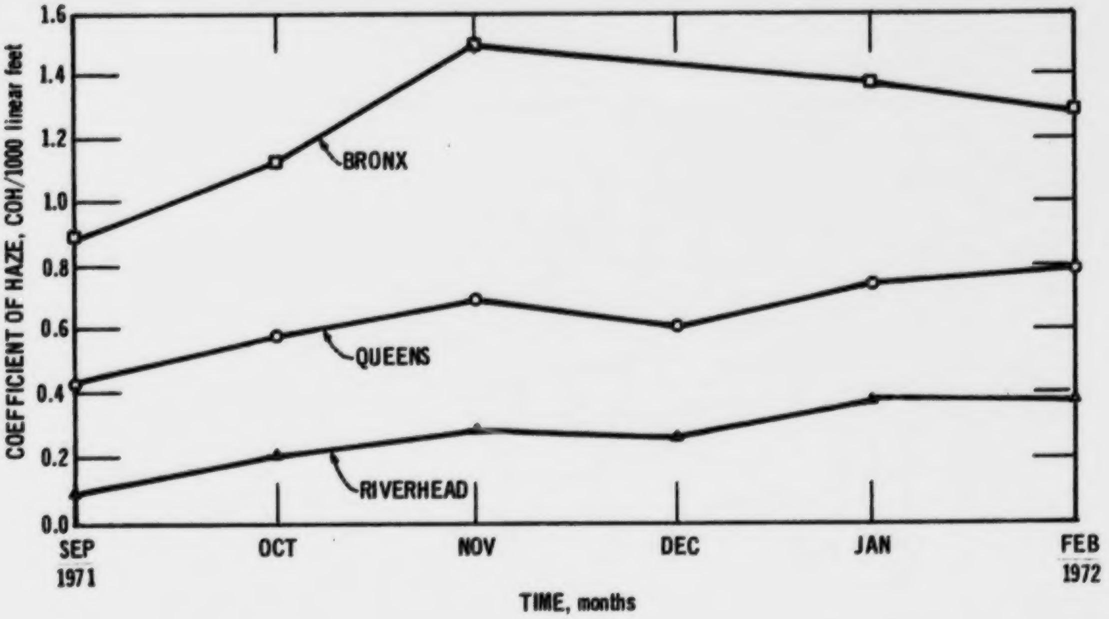

Figure 5.1.15. Coefficient of haze versus time in months, New York CHESS.

Bronx than in Queens or Riverhead (Figure 5.1.17). Both Queens and Bronx are characterized by monthly average cadmium values of about 0.1 mg/m$^2$/mo, while the levels in Riverhead are about 0.05 mg/m$^2$/mo. Zinc in monthly dustfall samples shows a gradient during most of the year, decreasing from Bronx, to Queens, to Riverhead (Figure 5.1.18). The respective average monthly zinc values in dustfall are 16, 8, and 4 mg/m$^2$/mo. A similar gradient is demonstrated by lead in monthly dustfall (Figure 5.1.19); mean monthly values over 17 months from Bronx, Queens, and Riverhead were 16, 10, and 2 mg/m$^2$/mo, respectively.

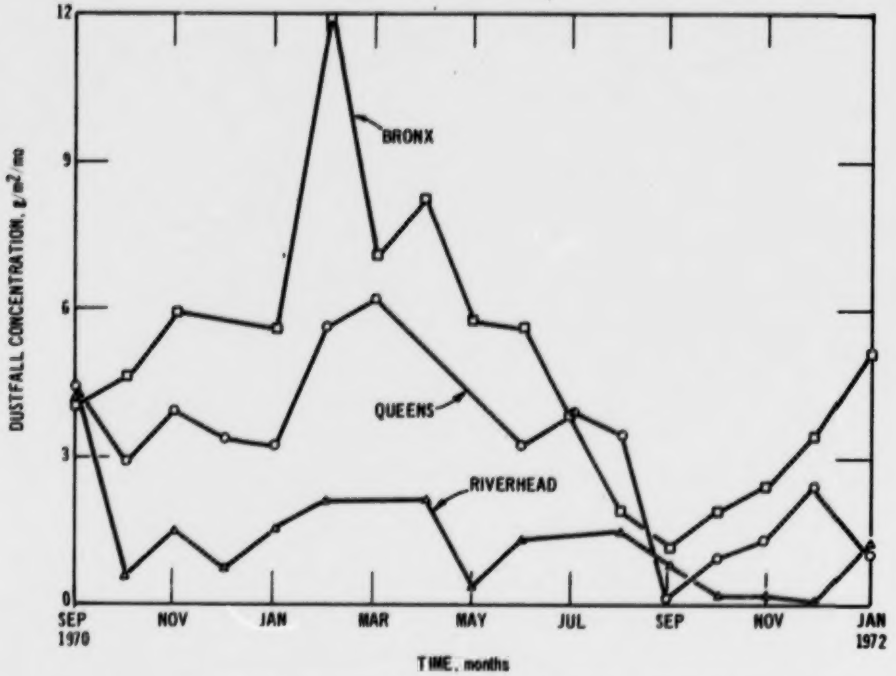

Figure 5.1.16. Dustfall concentration versus time in months, New York CHESS.

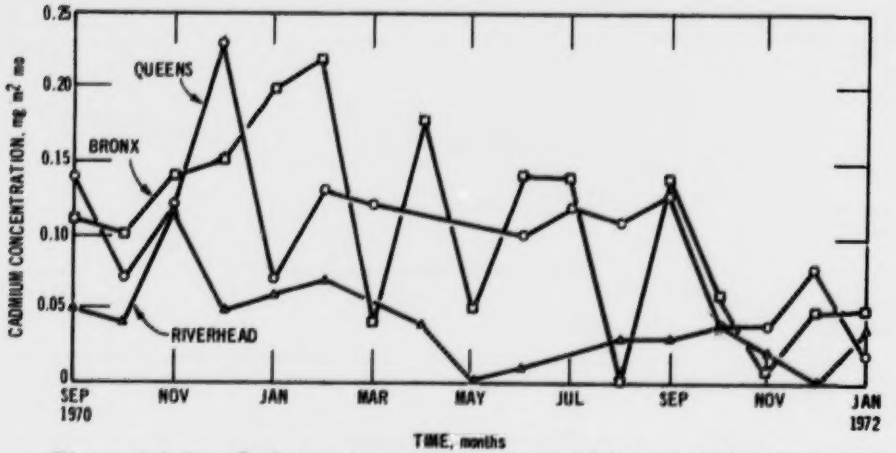

Figure 5.1.17. Cadmium concentration in dustfall versus time in months, New York CHESS.

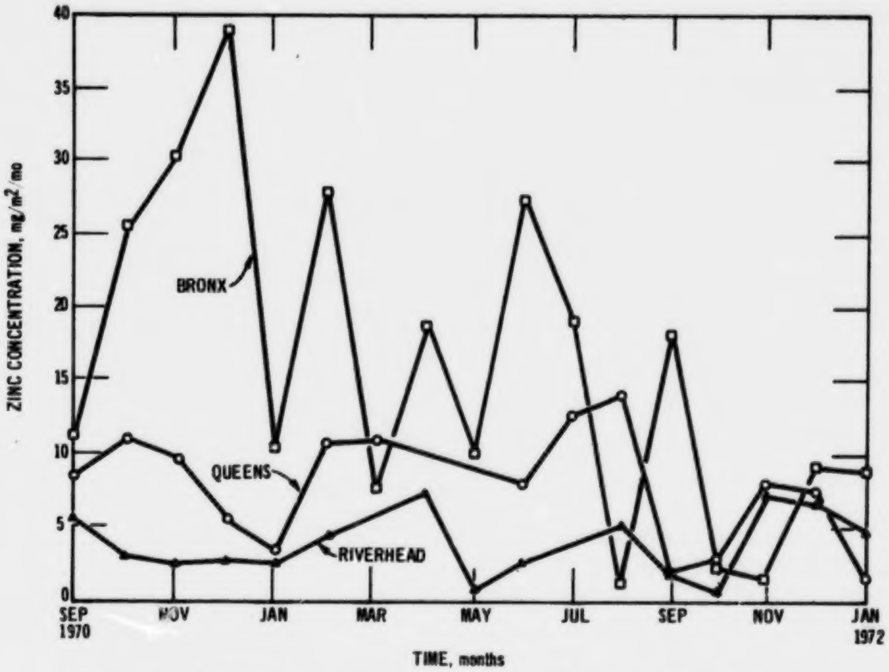

Figure 5.1.18. Zinc concentration in dustfall versus time in months, New York CHESS.

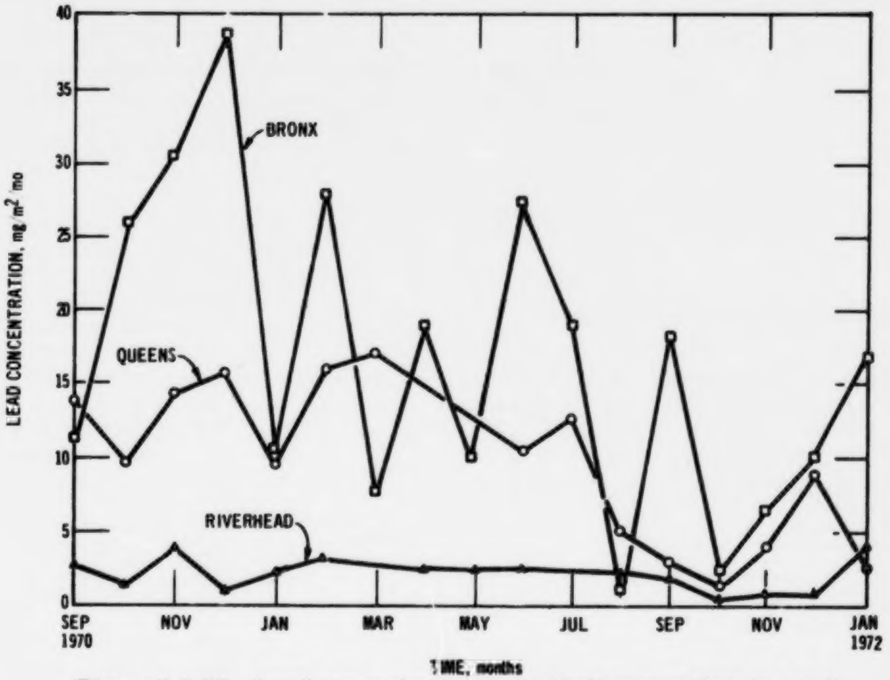

Figure 5.1.19. Lead concentration in dustfall versus time in months, New York CHESS.

## Interrelationships among Pollutants

### *Total Suspended Particulates, Suspended Nitrates, and Nitrogen Dioxide*

Comparisons of 24-hour integrated daily measurements for 1971 (Table 5.1.2) gave evidence that the weights of the nitrate fractions of total suspended particulate samples were consistently related to the total weights of the samples in the community with highest pollution levels (Bronx), but poorly correlated in the lowest pollution community (Riverhead). Nitrogen dioxide also correlated strongly with total suspended particulates in the high pollution community, but more weakly in the communities with less pollution. Nitrate levels showed weak correlation with nitrogen dioxide levels.

**Table 5.1.2. CORRELATION COEFFICIENTS FOR TOTAL SUSPENDED PARTICULATES, SUSPENDED NITRATES, AND NITROGEN DIOXIDE, NEW YORK CHESS, 1971**

| Community | Pollutant | Correlation coefficient TSP | SN | $NO_2$ |
|---|---|---|---|---|
| Bronx | TSP | 1.000 | 0.594 | 0.545 |
| | SN | 0.594 | 1.000 | 0.361 |
| | $NO_2$ | 0.545 | 0.361 | 1.000 |
| Queens | TSP | 1.000 | 0.430 | 0.374 |
| | SN | 0.430 | 1.000 | 0.190 |
| | $NO_2$ | 0.374 | 0.190 | 1.000 |
| Riverhead | TSP | 1.000 | 0.198 | 0.368 |
| | SN | 0.198 | 1.000 | 0.323 |
| | $NO_2$ | 0.368 | 0.323 | 1.000 |

### *Total and Respirable Suspended Particulates*

Comparisons of nine monthly arithmetic means for total and respirable suspended particulates indicated a rather consistent relationship. Ratios of respirable to total particulates decreased from the areas of low pollution to the area of high pollution (Figure 5.1.20).

### *Total Suspended Particulates, Suspended Sulfates, and Sulfur Dioxide*

Comparisons of 24-hour integrated daily measurements for 1971 (Table 5.1.3) indicated

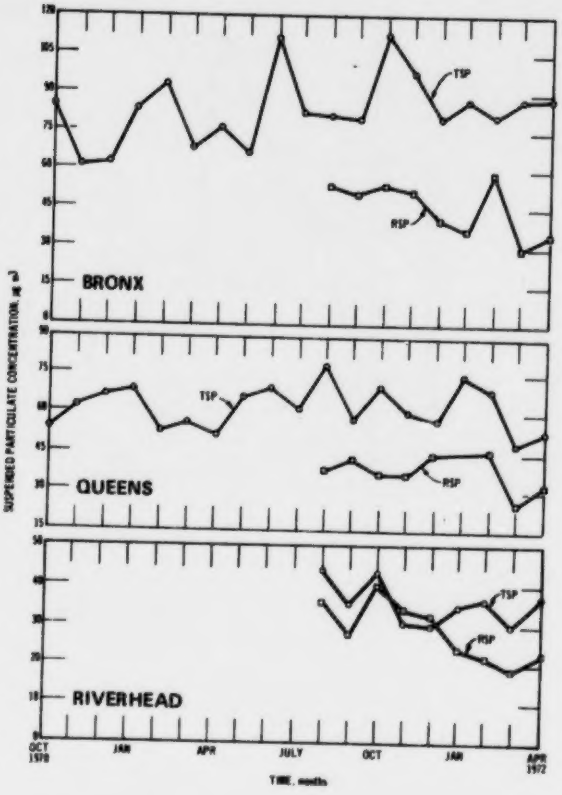

Figure 5.1.20. Total suspended particulate (high volume sampler) and respirable suspended particulate (cyclone) concentrations versus time in months, New York CHESS.

**Table 5.1.3. CORRELATION COEFFICIENTS FOR TOTAL SUSPENDED PARTICULATES, SUSPENDED SULFATES, AND SULFUR DIOXIDE, NEW YORK CHESS, 1971**

| Community | Pollutant | Correlation coefficient TSP | SS | $SO_2$ |
|---|---|---|---|---|
| Bronx | TSP | 1.000 | 0.615 | 0.150 |
| | SS | 0.615 | 1.000 | 0.130 |
| | $SO_2$ | 0.150 | 0.130 | 1.000 |
| Queens | TSP | 1.000 | 0.637 | 0.399 |
| | SS | 0.637 | 1.000 | 0.282 |
| | $SO_2$ | 0.399 | 0.282 | 1.000 |
| Riverhead | TSP | 1.000 | 0.772 | 0.315 |
| | SS | 0.772 | 1.000 | 0.282 |
| | $SO_2$ | 0.315 | 0.282 | 1.000 |

that the weights of the sulfate fractions of total suspended particulate samples were consistently related to the total weights of the samples. This correlation was strongest (0.772) in the community with lowest total suspended particulate levels (Riverhead) and weakest (0.615) in the community with highest levels (Bronx). Similar comparisons of atmospheric sulfur dioxide and concurrent suspended sulfates showed less significant correlations.

## Long-term Exposure Trends

Estimates of previous exposures to airborne pollutants, extending back to 1944 (Table 5.1.A.8), were derived from information provided by NYC-DAR, supplemented by recent CHESS measurements and limited data from the National Air Sampling Network (NASN). In general, the citywide estimates were based on 12 years of recorded data (1958-1970) for atmospheric sulfur dioxide concentrations and 14 years (1956-1970) of data for monthly dustfall. Because fuel consumption and emission control technology remained reasonably constant for several years prior to 1958, it was assumed that the ratio between dustfall and sulfur dioxide concentration also remained constant during those years. Therefore, sulfur dioxide concentrations were estimated on the basis of available dustfall measurements for 1956-1957. Because the fluctuations in concentrations of total suspended particulate and sulfur dioxide follow the same general pattern, total suspended particulate concentrations back to 1956 also were estimated. Measured values for suspended sulfates for 1956-1970 were available from the Manhattan 121st Street station, and these values were used as citywide values. Pollution estimates for Queens and the Bronx were based on measured values or on relationships with citywide data, as described in the following paragraphs.

Annual arithmetic mean dustfall values for New York City are shown in Figure 5.1.21. In addition to citywide averages, data are presented from Bronx, Queens, and Manhattan. The values shown for Bronx and Queens prior to 1960 are borough averages. Starting with 1969, dustfall for Queens and Bronx was collected at the present CHESS sites. For 1960-1969, values for Queens and Bronx in Table 5.1.A.8 were obtained by interpolation. Major decreases in dustfall levels occurred during the periods 1944-1952 and 1962-1969. For example, the citywide value in 1969 was only 14 percent of the 1944 value.

Long-term citywide trends for sulfur dioxide concentrations were determined by annual summarization of monthly data for 1958-1970. The citywide sulfur dioxide data for 1956-1957 were projected by graphically estimating the ratio of sulfur dioxide concentration to dustfall and multiplying the annual dustfall by the appropriate ratio. Citywide data for 1958-1970 were available from NYC-DAR. From 1965 to 1969, NYC-DAR measured sulfur dioxide in Queens and Bronx, providing an indication of average values for the boroughs. Prior to 1965, all values were estimated in the same manner as the 1956-1957 citywide values. Sulfur dioxide data for these areas were collected at the present CHESS sites after 1969. Most of the NYC-DAR measurements utilized the hydrogen perioxide technique; however, at some locations an electroconductivity method was used.

As indicated in Figure 5.1.22, dramatic reductions in average sulfur dioxide concentrations in New York City have occurred since 1964. However, the 1970 mean ambient levels still exceeded the National Annual Primary Air Quality Standard, even though they were approximately 66 percent lower than the 1964 values.

Annual arithmetic mean concentrations of total suspended particulate from 1956 to 1970 are shown in Figure 5.1.23. Citywide data for 1956-1957 were estimated by using the observed time-dependent ratio of total suspended particulate to dustfall. Citywide data indicate that suspended particulate concentrations increased from 1958 to 1963. Since 1963, suspended particulate concentrations have decreased by 63 percent; however, the 1970 mean annual ambient level of 105 $\mu g/m^3$ was still in excess of the National Annual Primary Ambient Air Quality Standard (75 $\mu g/m^3$). In both Bronx and Queens, annual values were estimated by multiplying the appropriate annual dustfall values by the citywide ratio of total suspended particulate to dustfall.

Suspended sulfate data obtained from the 121st Street sampling station in Manhattan are shown as citywide values in Figure 5.1.24. Citywide suspended sulfate data for 1959-1961 were obtained by interpolation. The observed annual ratios of suspended sulfate to dustfall for New York City were used to estimate the suspended sulfate levels in Queens and Bronx. These estimates indicate that suspended sulfate levels, which show a gradual decrease since 1960, are quite similar in Queens and Bronx.

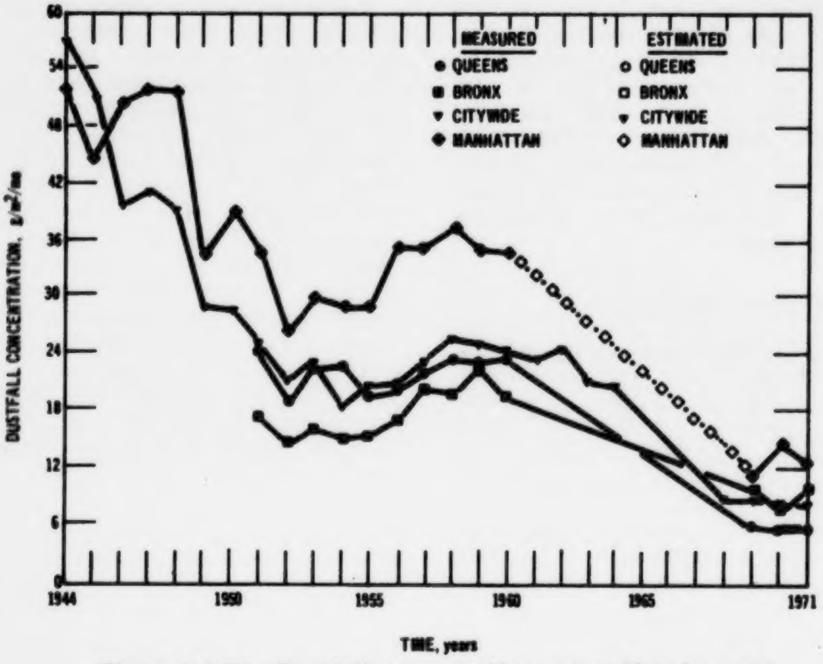

Figure 5.1.21. Dustfall concentration versus time in years for New York City.

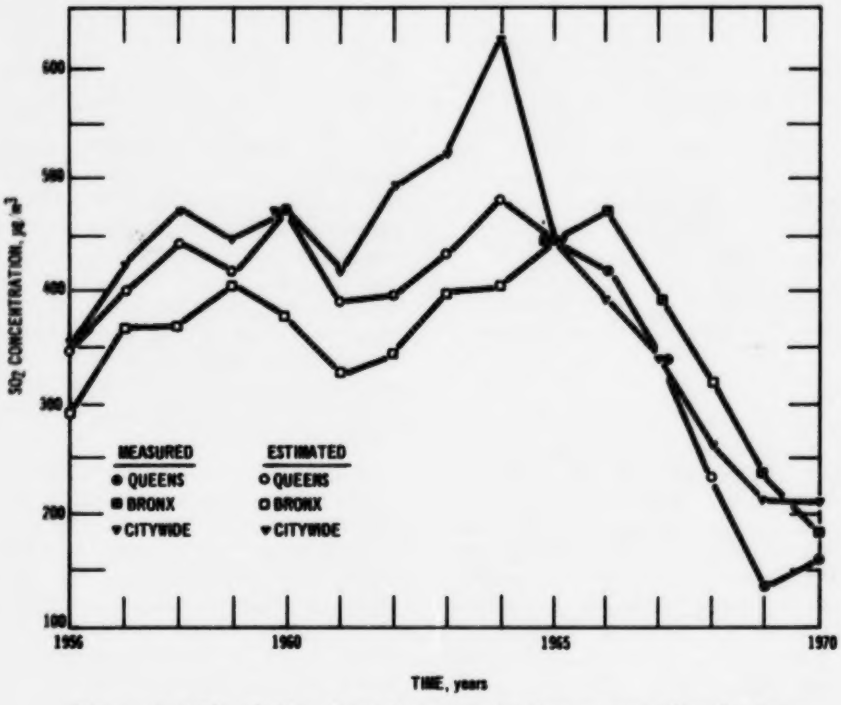

Figure 5.1.22. Sulfur dioxide concentration versus time in years for New York City.

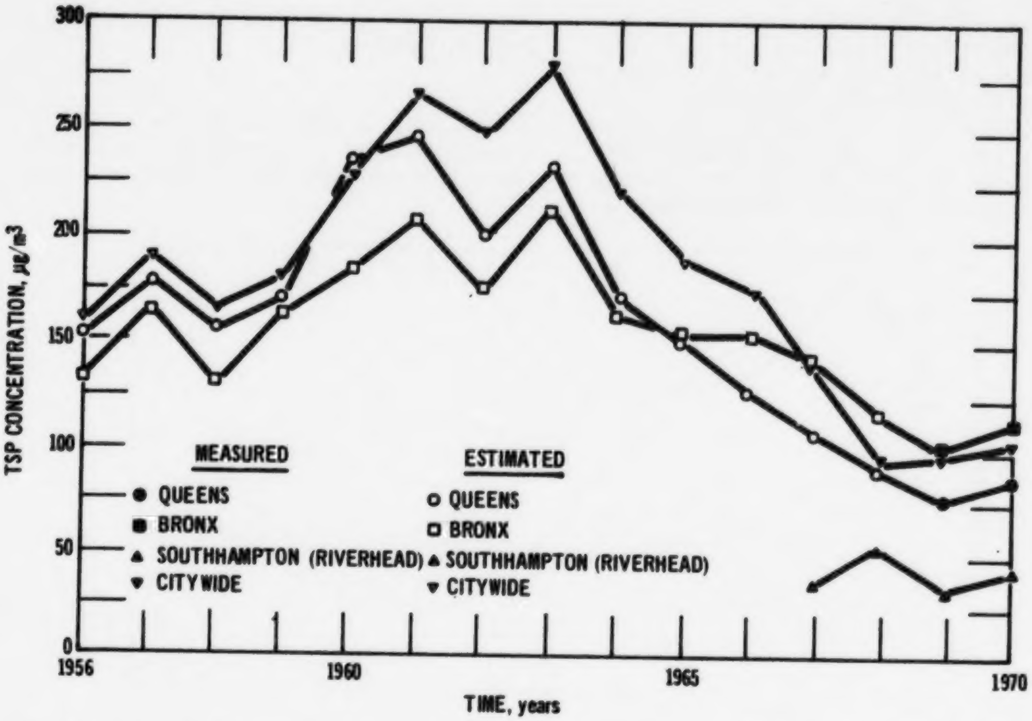

Figure 5.1.23. Total suspended particulate concentration versus time in years for New York City.

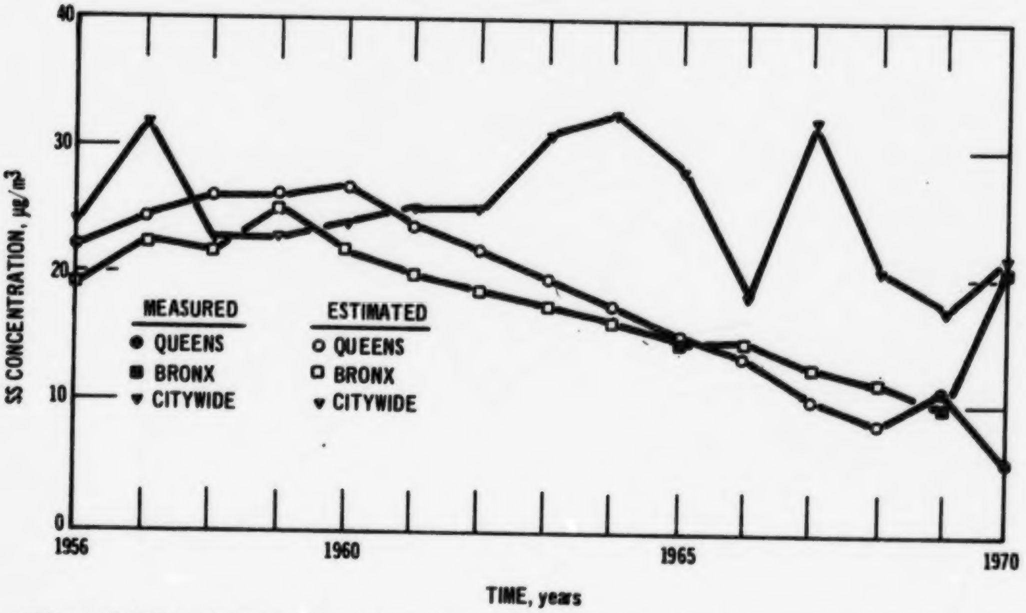

Figure 5.1.24. Suspended sulfate concentration versus time in years for New York City.

Annual NYC-DAR Manhattan dust count data from 1958 to 1971 are shown in Figure 5.1.25. The curve indicates that the dust count, related primarily to small particles, decreased by a factor of 5.6 from 1959 to 1966, then started an upward trend that continued through 1971. These recent increases indicate that, although total suspended particulate concentrations are decreasing, the quantities of respirable particulates could be increasing.

A summary of COH data for New York City is shown in Figure 5.1.26. Citywide annual COH values decreased from 2.5 in 1960 to 1.0 in 1971.

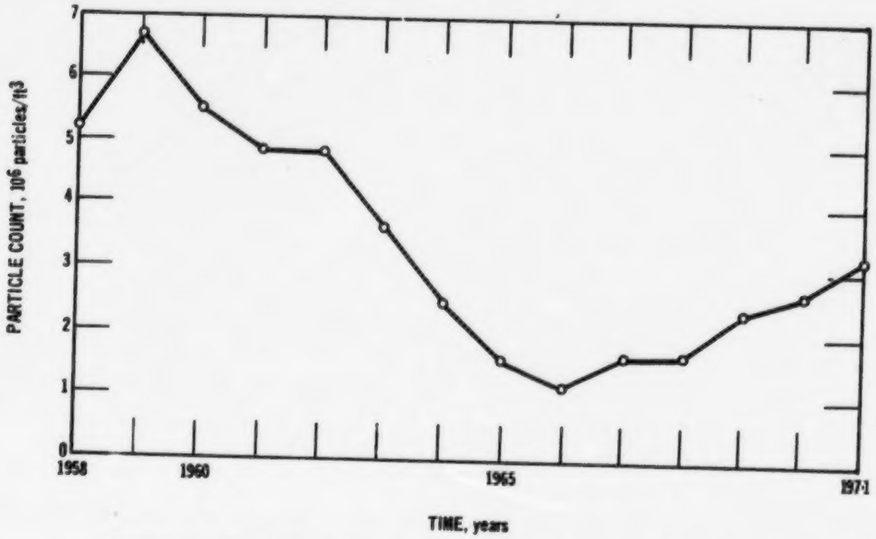

Figure 5.1.25. Dust count versus time in years for Manhattan.

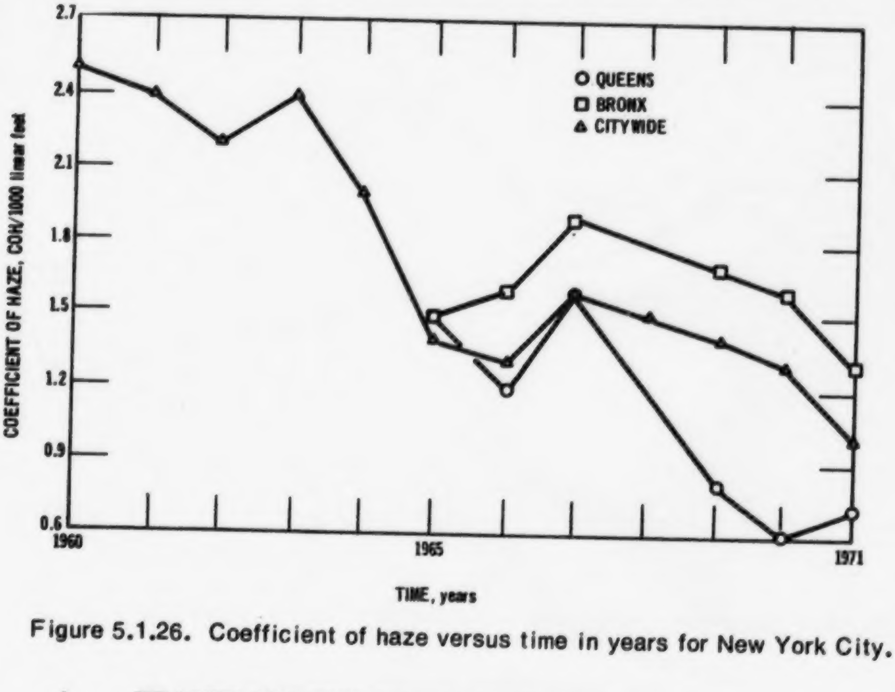

Figure 5.1.26. Coefficient of haze versus time in years for New York City.

## SUMMARY

CHESS data and available data from the New York City Department of Air Resources have been utilized to provide a comprehensive data base that is representative of historical air quality in the New York City metropolitan area. Pollutant levels for selected time periods are summarized in Table 5.1.4. Collection methods were compared for equivalency and correlation. Where data were not available, estimates were made based on the best available information. These data show a consistent improvement in air quality for all pollutants considered. Monthly sulfur dioxide concentrations follow a cyclic pattern, with peak concentrations occurring in January. No such pattern was exhibited for total suspended particulates, respirable suspended particulates, or suspended sulfates.

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Table 5.1.4. ARITHMETIC MEAN POLLUTION CONCENTRATIONS, NEW YORK CITY, 1956-1971

| Pollutant and location | Pollutant levels | | | | |
|---|---|---|---|---|---|
| | 1956-1959 | 1960-1963 | 1964-1967 | 1968-1970 | 1971 |
| Sulfur dioxide, $\mu g/m^3$ | | | | | |
| Citywide | 423 | 478 | 452 | 227 | 105 |
| Queens | 402 | 424 | 423 | 174 | 94 |
| Bronx | 359 | 362 | 445 | 247 | 107 |
| Total suspended particulates, $\mu g/m^3$ | | | | | |
| Citywide | 172 | 257 | 182 | 99 | 105 |
| Queens | 164 | 228 | 139 | 84 | 98 |
| Bronx | 147 | 195 | 152 | 108 | 105 |
| Suffolk Co. | – | – | – | 41 | 40 |
| Suspended sulfates, $\mu g/m^3$ | | | | | |
| Citywide | 25.5 | 26.4 | 28.2 | 20.5 | – |
| Queens | 24.8 | 23.3 | 14.6 | 8.6 | 10.1 |
| Bronx | 22.3 | 19.9 | 15.3 | 14.8 | 14.4 |
| Dustfall, $g/m^2/mo$ | | | | | |
| Citywide | 24.1 | 23.7 | 16.8 | 8.7 | 8.4 |
| Queens | 22.6 | 21.2 | 13.2 | 6.7 | 8.0 |
| Bronx | 20.3 | 18.1 | 13.9 | 9.7 | 7.9 |
| Suffolk Co. | – | – | – | 2.3 | – |

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### Table 5.1.A.1. DAILY SULFUR DIOXIDE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, SEPTEMBER 1970-MARCH 1972

| Community | Period of Observation | Observa-tions | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standard[b], % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arith-metic mean | Geo-metric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Riverhead | 9/70-12/70 | 73 | 0.0 | 130.6 | 21.3 | 13.7 | 1.11 | 1.4 | 7.0 | 13.5 | 24.5 | 44.5 | 56.2 | 124.4 | 2.7 | 0.0 |
|  | 1/71-3/71 | 78 | 1.2 | 125.4 | 30.2 | 18.4 | 1.07 | 3.9 | 10.2 | 17.7 | 36.1 | 76.3 | 90.2 | 123.9 | 7.7 | 0.0 |
|  | 4/71-6/71 | 87 | 0.0 | 113.6 | 21.2 | 14.2 | 1.07 | 2.2 | 8.7 | 15.1 | 24.7 | 44.8 | 60.5 | 79.9 | 1.1 | 0.0 |
|  | 7/71-9/71 | 84 | 0.0 | 52.4 | 10.3 | 6.0 | 1.25 | 0.5 | 3.3 | 7.6 | 12.0 | 21.6 | 27.6 | 49.2 | 0.0 | 0.0 |
|  | 10/71-12/71 | 76 | 1.7 | 144.9 | 30.1 | 20.8 | 0.94 | 6.3 | 14.5 | 23.8 | 36.8 | 61.2 | 81.0 | 111.0 | 6.6 | 0.0 |
|  | 1/71-12/71 | 325 | 0.0 | 144.9 | 22.6 | 13.3 | 1.19 | 2.4 | 8.0 | 15.1 | 25.8 | 53.0 | 73.0 | 111.1 | 3.7 | 0.0 |
|  | 1/72-3/72 | 83 | 1.1 | 146.5 | 39.7 | 26.9 | 1.03 | 6.1 | 21.2 | 32.1 | 51.6 | 88.8 | 96.6 | 113.2 | 13.3 | 0.0 |
| Queens | 9/70-12/70 | 67 | 0.0 | 230.7 | 65.4 | 45.6 | 1.16 | 10.2 | 33.5 | 57.2 | 86.4 | 119.8 | 160.5 | 205.4 | 31.3 | 0.0 |
|  | 1/71-3/71 | 73 | 4.4 | 287.1 | 57.6 | 37.1 | 0.97 | 11.7 | 18.4 | 33.5 | 86.2 | 118.5 | 153.5 | 231.0 | 32.9 | 0.0 |
|  | 4/71-6/71 | 90 | 0.0 | 142.8 | 52.7 | 37.0 | 1.04 | 9.9 | 25.5 | 47.7 | 66.6 | 108.2 | 122.8 | 142.3 | 22.2 | 0.0 |
|  | 7/71-9/71 | 84 | 1.6 | 107.4 | 31.9 | 21.8 | 1.00 | 5.5 | 13.6 | 29.0 | 42.1 | 70.9 | 81.0 | 107.1 | 7.1 | 0.0 |
|  | 10/71-12/71 | 84 | 0.4 | 246.9 | 59.2 | 43.7 | 0.97 | 11.1 | 38.3 | 53.9 | 71.8 | 100.2 | 114.8 | 202.2 | 23.8 | 0.0 |
|  | 1/71-12/71 | 331 | 0.0 | 287.1 | 50.1 | 33.7 | 1.03 | 8.0 | 22.7 | 38.5 | 63.9 | 104.1 | 121.0 | 190.2 | 21.1 | 0.0 |
|  | 1/72-3/72 | 85 | 0.0 | 172.8 | 59.6 | 42.9 | 1.09 | 11.3 | 39.1 | 56.2 | 71.3 | 126.6 | 146.8 | 168.0 | 21.2 | 0.0 |
| Bronx | 9/70-12/70 | 67 | 8.7 | 268.1 | 83.9 | 60.1 | 0.88 | 19.7 | 37.0 | 63.5 | 107.2 | 177.7 | 196.1 | 255.1 | 41.8 | 0.0 |
|  | 1/71-3/71 | 73 | 0.0 | 349.4 | 65.4 | 36.1 | 1.22 | 5.7 | 15.5 | 35.0 | 73.6 | 162.7 | 209.1 | 280.0 | 30.1 | 0.0 |
|  | 4/71-6/71 | 89 | 0.0 | 219.2 | 38.9 | 16.5 | 1.54 | 1.7 | 5.9 | 19.2 | 42.1 | 104.2 | 135.2 | 219.1 | 18.0 | 0.0 |
|  | 7/71-9/71 | 77 | 0.0 | 122.8 | 27.5 | 14.8 | 1.35 | 1.6 | 7.7 | 20.8 | 40.0 | 58.1 | 86.0 | 98.6 | 6.5 | 0.0 |
|  | 10/71-12/71 | 78 | 10.4 | 204.4 | 70.1 | 57.1 | 0.67 | 20.4 | 41.6 | 60.7 | 86.6 | 133.4 | 156.4 | 199.3 | 35.9 | 0.0 |
|  | 1/71-12/71 | 317 | 0.0 | 349.4 | 49.9 | 26.2 | 1.37 | 3.0 | 16.1 | 30.9 | 59.1 | 124.1 | 152.7 | 219.2 | 22.4 | 0.0 |
|  | 1/72-3/72 | 86 | 0.0 | 272.9 | 85.2 | 60.5 | 1.04 | 17.2 | 50.4 | 75.3 | 104.8 | 177.3 | 197.9 | 239.2 | 46.5 | 0.0 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1- National Annual Primary Air Quality Standard, 80 μg/m³.
Standard 2- National Daily Primary Air Quality Standard, 365 μg/m³.

### Table 5.1.A.2. DAILY TOTAL SUSPENDED PARTICULATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, SEPTEMBER 1970-MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m$^3$ | | | | Geometric standard deviation | Frequency distribution[a] | | | | | | | Observations exceeding standard,[b] % | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Minimum | Maximum | Arithmetic mean | Geometric mean | | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Riverhead | 9/70-12/70 | 77 | 10.7 | 97.1 | 34.9 | 31.2 | 0.48 | 17.9 | 22.7 | 31.5 | 39.1 | 58.4 | 63.8 | 95.6 | 3.9 | 0.0 |
| | 1/71- 3/71 | 82 | 4.8 | 134.4 | 33.5 | 28.7 | 0.55 | 15.4 | 22.1 | 28.8 | 36.3 | 56.3 | 60.9 | 133.3 | 3.7 | 0.0 |
| | 4/71- 6/71 | 89 | 9.4 | 91.1 | 32.5 | 29.4 | 0.46 | 15.8 | 23.5 | 29.4 | 37.3 | 52.9 | 61.5 | 70.2 | 1.1 | 0.0 |
| | 7/71- 9/71 | 80 | 7.8 | 104.2 | 36.8 | 33.3 | 0.45 | 19.4 | 26.9 | 34.0 | 40.2 | 57.5 | 66.9 | 99.7 | 3.3 | 0.0 |
| | 10/71-12/71 | 85 | 10.5 | 89.3 | 34.6 | 31.2 | 0.45 | 18.7 | 24.1 | 29.9 | 40.9 | 53.5 | 71.7 | 84.9 | 4.7 | 0.0 |
| | 1/71-12/71 | 346 | 4.8 | 134.4 | 34.4 | 30.6 | 0.48 | 17.3 | 24.5 | 29.9 | 39.4 | 55.3 | 64.0 | 96.3 | 3.2 | 0.0 |
| | 1/72- 3/72 | 85 | 12.7 | 105.4 | 34.7 | 32.8 | 0.33 | 22.7 | 28.1 | 33.0 | 37.4 | 48.5 | 53.1 | 76.8 | 1.2 | 0.0 |
| Queens | 9/70-12/70 | 65 | 23.3 | 189.5 | 61.5 | 55.6 | 0.44 | 32.4 | 42.6 | 56.1 | 70.1 | 93.2 | 121.8 | 163.0 | 24.6 | 0.0 |
| | 1/71- 3/71 | 83 | 12.5 | 156.2 | 59.3 | 54.8 | 0.41 | 37.4 | 46.8 | 55.3 | 63.5 | 89.7 | 109.4 | 140.3 | 15.7 | 0.0 |
| | 4/71- 6/71 | 90 | 23.5 | 193.4 | 63.0 | 57.9 | 0.41 | 35.8 | 46.6 | 57.1 | 71.5 | 102.3 | 110.4 | 132.0 | 26.7 | 0.0 |
| | 7/71- 9/71 | 82 | 22.5 | 151.5 | 66.5 | 61.7 | 0.39 | 40.0 | 50.8 | 60.1 | 76.4 | 100.9 | 127.8 | 142.5 | 32.9 | 0.0 |
| | 10/71-12/71 | 85 | 24.9 | 172.9 | 63.7 | 57.9 | 0.43 | 35.2 | 45.3 | 57.7 | 70.3 | 108.0 | 120.3 | 168.7 | 23.5 | 0.0 |
| | 1/71-12/71 | 340 | 12.5 | 193.4 | 63.1 | 58.0 | 0.41 | 36.2 | 46.7 | 57.1 | 69.1 | 101.3 | 115.5 | 156.2 | 24.7 | 0.0 |
| | 1/72- 3/72 | 78 | 26.2 | 212.7 | 64.9 | 59.1 | 0.42 | 35.5 | 49.1 | 56.0 | 70.5 | 98.5 | 119.2 | 193.6 | 23.1 | 0.0 |
| Bronx | 9/70-12/70 | 73 | 15.5 | 216.4 | 68.5 | 59.7 | 0.52 | 33.3 | 45.4 | 57.3 | 75.0 | 117.1 | 153.7 | 191.4 | 30.1 | 0.0 |
| | 1/71- 3/71 | 77 | 33.8 | 187.7 | 81.6 | 75.6 | 0.39 | 46.3 | 58.0 | 75.3 | 95.2 | 120.8 | 150.1 | 174.3 | 50.6 | 0.0 |
| | 4/71- 6/71 | 90 | 23.4 | 217.4 | 84.5 | 74.7 | 0.51 | 39.6 | 57.0 | 76.9 | 100.8 | 128.4 | 175.3 | 209.9 | 53.3 | 0.0 |
| | 7/71- 9/71 | 87 | 7.1 | 210.5 | 81.5 | 74.2 | 0.47 | 41.4 | 61.2 | 77.7 | 91.1 | 117.9 | 131.1 | 189.3 | 56.3 | 0.0 |
| | 10/71-12/71 | 83 | 34.0 | 274.7 | 98.5 | 88.1 | 0.46 | 49.9 | 70.8 | 84.0 | 105.1 | 161.1 | 177.8 | 265.5 | 62.7 | 2.4 |
| | 1/71-12/71 | 337 | 7.1 | 274.7 | 86.5 | 77.8 | 0.47 | 43.7 | 60.1 | 78.0 | 96.9 | 138.0 | 168.7 | 232.9 | 55.8 | 0.6 |
| | 1/72- 3/72 | 82 | 30.0 | 196.7 | 86.2 | 80.0 | 0.39 | 50.2 | 68.2 | 76.5 | 98.6 | 139.0 | 148.9 | 191.0 | 53.7 | 0.0 |

[a] Concentrations (μg/m$^3$) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 75 μg/m$^3$.
Standard 2 - National Daily Primary Air Quality Standard, 260 μg/m$^3$.

## Table 5.1.A.3. DAILY SUSPENDED SULFATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, SEPTEMBER 1970-MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 |
| Riverhead | 9/70-12/70 | 77 | 0.0 | 47.8 | 12.4 | 9.6 | 0.88 | 2.5 | 8.7 | 11.3 | 15.2 | 22.8 | 26.1 | 34.6 |
|  | 1/71- 3/71 | 82 | 2.1 | 51.4 | 10.4 | 8.8 | 0.56 | 4.8 | 6.4 | 8.7 | 11.5 | 17.9 | 20.9 | 45.7 |
|  | 4/71- 6/71 | 89 | 1.1 | 31.6 | 7.9 | 6.5 | 0.65 | 3.0 | 5.0 | 6.0 | 8.7 | 16.0 | 17.2 | 26.7 |
|  | 7/71- 9/71 | 90 | 3.0 | 41.9 | 11.2 | 9.2 | 0.61 | 4.4 | 6.2 | 9.3 | 12.1 | 21.6 | 27.3 | 41.0 |
|  | 10/71-12/71 | 85 | 3.0 | 30.3 | 11.3 | 10.2 | 0.45 | 5.9 | 7.6 | 10.1 | 12.8 | 19.0 | 22.2 | 28.0 |
|  | 1/71-12/71 | 346 | 1.1 | 51.4 | 10.2 | 8.5 | 0.60 | 4.2 | 6.2 | 8.5 | 11.5 | 18.4 | 22.9 | 36.7 |
|  | 1/72- 3/72 | 85 | 4.9 | 35.4 | 12.0 | 10.9 | 0.42 | 7.2 | 8.6 | 10.1 | 12.3 | 21.6 | 24.2 | 31.6 |
| Queens | 9/70-12/70 | 66 | 0.8 | 28.0 | 14.5 | 12.8 | 0.61 | 8.0 | 11.6 | 14.1 | 17.3 | 22.4 | 24.4 | 27.0 |
|  | 1/71- 3/71 | 83 | 5.4 | 39.3 | 13.7 | 12.7 | 0.39 | 8.4 | 10.0 | 11.8 | 14.8 | 19.7 | 27.5 | 36.9 |
|  | 4/71- 6/71 | 90 | 1.9 | 36.4 | 10.8 | 9.6 | 0.49 | 6.0 | 7.2 | 9.1 | 11.8 | 17.2 | 20.6 | 30.9 |
|  | 7/71- 9/71 | 82 | 2.8 | 36.5 | 14.7 | 12.6 | 0.57 | 5.8 | 9.4 | 12.4 | 17.4 | 27.2 | 31.9 | 36.1 |
|  | 10/71-12/71 | 86 | 2.6 | 33.8 | 13.5 | 12.1 | 0.47 | 7.1 | 9.2 | 12.2 | 15.0 | 22.4 | 29.9 | 32.2 |
|  | 1/71-12/71 | 341 | 1.9 | 39.3 | 13.2 | 11.6 | 0.50 | 6.5 | 8.9 | 11.3 | 14.9 | 21.9 | 29.2 | 36.2 |
|  | 1/72- 3/72 | 78 | 6.9 | 38.2 | 14.1 | 13.2 | 0.35 | 8.8 | 10.8 | 12.4 | 14.9 | 21.8 | 24.7 | 32.8 |
| Bronx | 9/70-12/70 | 73 | 0.0 | 35.7 | 13.5 | 11.9 | 0.65 | 5.3 | 9.7 | 13.6 | 15.8 | 21.5 | 25.8 | 33.1 |
|  | 1/71- 3/71 | 77 | 5.0 | 35.1 | 14.7 | 13.5 | 0.42 | 8.1 | 10.9 | 14.1 | 16.4 | 22.6 | 25.3 | 34.7 |
|  | 4/71- 6/71 | 90 | 2.3 | 41.6 | 12.0 | 10.1 | 0.59 | 4.9 | 7.4 | 9.8 | 13.1 | 22.2 | 28.7 | 38.5 |
|  | 7/71- 9/71 | 87 | 3.7 | 52.9 | 16.2 | 13.6 | 0.59 | 6.3 | 9.2 | 14.2 | 18.9 | 31.6 | 34.2 | 51.4 |
|  | 10/71-12/71 | 84 | 5.7 | 34.1 | 14.7 | 13.4 | 0.43 | 7.6 | 10.7 | 12.8 | 16.2 | 25.0 | 29.8 | 32.4 |
|  | 1/71-12/71 | 338 | 2.3 | 52.9 | 14.3 | 12.5 | 0.53 | 6.7 | 9.4 | 12.5 | 15.9 | 24.7 | 31.6 | 39.4 |
|  | 1/72- 3/72 | 82 | 6.1 | 32.7 | 14.0 | 13.1 | 0.36 | 8.4 | 10.3 | 13.1 | 15.8 | 20.3 | 23.3 | 29.4 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

## Table 5.1.A.4. DAILY SUSPENDED NITRATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, SEPTEMBER 1970-MARCH 1972

| Community | Period of observation | Observa-tions | Concentration, μg/m3 |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arith-metic mean | Geo-metric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 |
| Riverhead | 9/70-12/70 | 77 | 0.2 | 3.4 | 1.4 | 1.2 | 0.58 | 0.6 | 0.9 | 1.3 | 1.7 | 2.6 | 2.9 | 3.3 |
|  | 1/71- 3/71 | 82 | 0.2 | 6.4 | 1.3 | 1.0 | 0.64 | 0.5 | 0.7 | 1.2 | 1.4 | 2.0 | 2.7 | 4.7 |
|  | 4/71- 6/71 | 39 | 0.0 | 3.6 | 1.0 | 0.8 | 0.71 | 0.3 | 0.6 | 0.8 | 1.0 | 2.0 | 2.3 | 3.5 |
|  | 7/71- 9/71 | 90 | 0.0 | 5.8 | 2.1 | 1.6 | 0.93 | 0.4 | 1.4 | 1.9 | 2.5 | 3.8 | 4.8 | 5.6 |
|  | 10/71-12/71 | 85 | 0.1 | 8.7 | 3.1 | 2.5 | 0.77 | 0.9 | 2.0 | 2.8 | 3.7 | 5.8 | 6.5 | 7.9 |
|  | 1/71-12/71 | 346 | 0.0 | 8.7 | 1.9 | 1.3 | 0.89 | 0.4 | 0.8 | 1.4 | 2.2 | 3.8 | 5.0 | 6.6 |
|  | 1/72- 3/72 | 85 | 0.1 | 9.3 | 2.6 | 1.9 | 0.94 | 0.6 | 1.6 | 2.1 | 3.0 | 5.0 | 6.2 | 7.7 |
| Queens | 9/70-12/70 | 66 | 0.3 | 5.5 | 1.7 | 1.5 | 0.58 | 0.7 | 1.2 | 1.7 | 2.0 | 2.8 | 3.0 | 5.5 |
|  | 1/71- 3/71 | 83 | 0.2 | 5.6 | 1.7 | 1.4 | 0.60 | 0.8 | 1.1 | 1.4 | 1.9 | 3.2 | 3.4 | 4.8 |
|  | 4/71- 6/71 | 90 | 0.4 | 11.3 | 2.6 | 2.1 | 0.73 | 0.7 | 1.4 | 2.4 | 3.2 | 5.1 | 6.1 | 8.3 |
|  | 7/71- 9/71 | 82 | 0.0 | 14.7 | 4.6 | 3.9 | 0.67 | 1.8 | 2.9 | 4.1 | 5.7 | 8.0 | 9.9 | 12.6 |
|  | 10/71-12/71 | 86 | 0.4 | 23.5 | 5.2 | 4.0 | 0.77 | 1.5 | 2.7 | 4.1 | 5.7 | 10.6 | 13.8 | 20.3 |
|  | 1/71-12/71 | 341 | 0.0 | 23.5 | 3.5 | 2.6 | 0.82 | 0.9 | 1.6 | 2.6 | 4.1 | 6.8 | 8.9 | 14.8 |
|  | 1/72- 3/72 | 78 | 0.2 | 12.2 | 4.0 | 3.2 | 0.70 | 1.4 | 2.3 | 3.2 | 4.5 | 8.4 | 9.4 | 12.2 |
| Bronx | 9/70-12/70 | 73 | 0.1 | 4.7 | 1.6 | 1.4 | 0.61 | 0.8 | 1.0 | 1.4 | 1.9 | 2.9 | 3.1 | 4.5 |
|  | 1/71- 3/71 | 77 | 0.0 | 6.1 | 1.9 | 1.6 | 0.66 | 0.8 | 1.1 | 1.5 | 2.1 | 3.9 | 4.6 | 5.9 |
|  | 4/71- 6/71 | 90 | 0.4 | 20.4 | 4.2 | 2.9 | 0.93 | 0.8 | 1.8 | 3.0 | 5.1 | 9.0 | 12.6 | 15.9 |
|  | 7/71- 9/71 | 87 | 0.5 | 13.7 | 4.8 | 4.1 | 0.60 | 2.0 | 3.3 | 4.1 | 5.6 | 8.6 | 9.9 | 13.2 |
|  | 10/71-12/71 | 84 | 0.2 | 18.3 | 5.1 | 3.8 | 0.82 | 1.4 | 2.6 | 4.0 | 5.6 | 10.4 | 13.3 | 17.6 |
|  | 1/71-12/71 | 338 | 0.0 | 20.4 | 4.1 | 2.9 | 0.85 | 1.0 | 1.4 | 3.2 | 4.7 | 8.1 | 11.3 | 16.6 |
|  | 1/72- 3/72 | 82 | 0.0 | 15.7 | 3.7 | 2.4 | 1.19 | 0.5 | 2.0 | 3.0 | 4.2 | 6.9 | 10.1 | 14.7 |

[a] Concentrations ($\mu g/m^3$) distributed by cumulative frequency of occurrence (percent).

### Table 5.1.A.5. DAILY RESPIRABLE SUSPENDED PARTICULATE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, JULY 1971-MARCH 1972

| Community | Period of observation | Observations | Concentration, µg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 |
| Riverhead | 7/71- 9/71 | 63 | 8.4 | 92.6 | 31.9 | 27.4 | 0.55 | 15.6 | 20.1 | 25.5 | 35.3 | 57.6 | 67.5 | 91.8 |
|  | 10/71-12/71 | 71 | 0.0 | 93.1 | 35.0 | 29.8 | 0.65 | 13.2 | 20.9 | 31.3 | 39.0 | 71.7 | 78.8 | 85.9 |
|  | 1/72- 3/72 | 73 | 0.0 | 75.7 | 21.7 | 17.5 | 0.81 | 6.0 | 15.1 | 21.1 | 26.9 | 35.2 | 39.2 | 60.1 |
| Queens | 7/71- 9/71 | 34 | 6.3 | 68.8 | 41.4 | 37.6 | 0.52 | 22.7 | 34.5 | 43.7 | 48.6 | 58.7 | 66.0 | 68.2 |
|  | 10/71-12/71 | 31 | 2.4 | 96.3 | 37.2 | 28.1 | 0.85 | 11.1 | 19.8 | 36.5 | 45.8 | 73.5 | 81.6 | 92.6 |
|  | 1/72- 3/72 | 36 | 2.9 | 92.5 | 29.4 | 24.1 | 0.73 | 12.4 | 20.2 | 28.0 | 31.6 | 47.5 | 62.4 | 82.0 |
| Bronx | 7/71- 9/71 | 54 | 10.1 | 88.7 | 52.9 | 48.5 | 0.46 | 24.6 | 41.0 | 58.4 | 65.2 | 73.3 | 82.0 | 86.7 |
|  | 10/71-12/71 | 69 | 4.1 | 195.8 | 48.6 | 38.7 | 0.69 | 19.8 | 28.3 | 40.4 | 50.0 | 84.0 | 103.9 | 193.0 |
|  | 1/72- 3/72 | 76 | 0.8 | 329.8 | 41.5 | 28.4 | 1.04 | 9.0 | 25.1 | 35.3 | 50.0 | 70.9 | 78.1 | 148.3 |

[a] Concentrations (µg/m³) distributed by cumulative frequency of occurrence (percent).

## Table 5.1.A.6. DAILY NITROGEN DIOXIDE CONCENTRATIONS AND FREQUENCY DISTRIBUTIONS FOR NEW YORK CHESS COMMUNITIES, JANUARY 1971-MARCH 1972

| Community | Period of observation | Observations | Concentration, μg/m³ |  |  |  | Geometric standard deviation | Frequency distribution[a] |  |  |  |  |  |  | Observations exceeding standards,[b] % |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Minimum | Maximum | Arithmetic mean | Geometric mean |  | 10 | 30 | 50 | 70 | 90 | 95 | 99 | Std. 1 | Std. 2 |
| Riverhead | 1/71- 3/71 | 74 | 0.0 | 69.2 | 24.7 | 21.2 | 0.61 | 8.2 | 15.0 | 20.0 | 30.0 | 49.3 | 55.0 | 64.5 | 0.0 | 0.0 |
|  | 4/71- 6/71 | 85 | 0.0 | 37.7 | 13.8 | 11.7 | 0.64 | 5.1 | 8.7 | 12.3 | 16.3 | 25.6 | 28.9 | 33.5 | 0.0 | 0.0 |
|  | 7/71- 9/71 | 87 | 3.7 | 76.5 | 17.2 | 14.6 | 0.58 | 6.8 | 10.5 | 15.3 | 21.3 | 28.7 | 33.8 | 45.0 | 0.0 | 0.0 |
|  | 10/71-12/71 | 74 | 5.6 | 86.7 | 29.0 | 23.4 | 0.68 | 9.2 | 15.1 | 25.5 | 35.2 | 56.2 | 67.7 | 81.2 | 0.0 | 0.0 |
|  | 1/71-12/71 | 320 | 0.0 | 86.7 | 20.7 | 16.7 | 0.69 | 6.6 | 11.5 | 16.7 | 24.1 | 39.3 | 53.6 | 70.6 | 0.0 | 0.0 |
|  | 1/72- 3/72 | 72 | 2.0 | 106.4 | 26.7 | 21.4 | 0.70 | 9.0 | 15.6 | 22.2 | 30.6 | 51.7 | 55.2 | 81.2 | 1.4 | 0.0 |
| Queens | 1/71- 3/71 | 74 | 7.7 | 14.6 | 62.4 | 56.6 | 0.49 | 33.4 | 48.8 | 60.1 | 75.7 | 100.8 | 106.4 | 111.7 | 10.8 | 0.0 |
|  | 4/71- 6/71 | 87 | 5.6 | 28.7 | 47.3 | 41.0 | 0.57 | 22.0 | 30.1 | 45.9 | 57.4 | 78.9 | 84.5 | 122.7 | 3.4 | 0.0 |
|  | 7/71- 9/71 | 91 | 11.2 | 143.4 | 55.8 | 47.9 | 0.57 | 22.0 | 33.3 | 50.6 | 69.7 | 95.3 | 118.6 | 140.4 | 8.8 | 0.0 |
|  | 10/71-12/71 | 81 | 4.9 | 142.5 | 64.7 | 57.4 | 0.54 | 31.4 | 46.5 | 60.4 | 76.3 | 97.7 | 122.8 | 138.8 | 9.9 | 0.0 |
|  | 1/71-12/71 | 333 | 4.9 | 143.4 | 57.2 | 49.9 | 0.56 | 23.6 | 41.1 | 54.0 | 69.8 | 94.3 | 108.8 | 137.8 | 8.1 | 0.0 |
|  | 1/72- 3/72 | 87 | 17.9 | 118.7 | 59.6 | 55.4 | 0.40 | 34.2 | 47.9 | 60.8 | 67.3 | 84.3 | 98 7 | 117.6 | 5.7 | 0.0 |
| Bronx | 1/71- 3/71 | 73 | 0.0 | 136.3 | 65.7 | 55.9 | 0.70 | 19.2 | 51.1 | 67.3 | 82.1 | 112.3 | 117.7 | 132.4 | 13.7 | 0.0 |
|  | 4/71- 6/71 | 89 | 8.3 | 169.9 | 61.7 | 55.8 | 0.49 | 32.0 | 45.3 | 59.3 | 71.2 | 96.3 | 105.1 | 127.9 | 7.9 | 0.0 |
|  | 7/71- 9/71 | 87 | 4.4 | 177.5 | 69.6 | 62.1 | 0.53 | 33.7 | 53.3 | 65.1 | 80.1 | 111.3 | 130.2 | 171.4 | 14.9 | 0.0 |
|  | 10/71-12/71 | 81 | 12.8 | 190.2 | 73.9 | 67.1 | 0.46 | 41.1 | 52.7 | 68.8 | 81.2 | 115.3 | 138.9 | 185.5 | 16.0 | 0.0 |
|  | 1/71-12/71 | 330 | 0.0 | 190.2 | 67.7 | 60.1 | 0.55 | 32.4 | 51.6 | 64.8 | 77.7 | 107.9 | 124.9 | 170.2 | 13.0 | 0.0 |
|  | 1/72- 3/72 | 81 | 1.4 | 681.9 | 72.7 | 59.6 | 0.67 | 37.9 | 52.3 | 65.5 | 78.3 | 99.4 | 109.7 | 229.7 | 9.9 | 1.2 |

[a] Concentrations (μg/m³) distributed by cumulative frequency of occurrence (percent).

[b] Standard 1 - National Annual Primary Air Quality Standard, 100 μg/m³.
Standard 2 - National Daily Primary Air Quality Standard, 250 μg/m³.

## Table 5.1.A.7. QUARTERLY ARITHMETIC MEAN POLLUTANT CONCENTRATIONS IN NEW YORK CITY CHESS COMMUNITIES

($\mu g/m^3$)

| Pollutant and location | Time, quarter of year | | | | | |
|---|---|---|---|---|---|---|
| | 4(1970) | 1(1971) | 2(1971) | 3(1971) | 4(1971) | 1(1972) |
| Sulfur dioxide | | | | | | |
| Riverhead | 20.7 | 32.2 | 21.5 | 10.1 | 32.0 | 40.4 |
| Queens | 63.1 | 60.9 | 53.8 | 31.7 | 60.3 | 63.0 |
| Bronx | 80.7 | 74.5 | 41.1 | 26.7 | 70.7 | 88.7 |
| Nitrogen dioxide | | | | | | |
| Riverhead | 45.5 | 40.6 | 13.8 | 17.2 | 30.7 | 27.7 |
| Queens | 102.2 | 63.1 | 47.8 | 55.9 | 64.9 | 59.5 |
| Bronx | 118.9 | 70.9 | 61.8 | 70.2 | 74.2 | 73.8 |
| Total suspended particulates | | | | | | |
| Riverhead | 35.1 | 33.5 | 32.6 | 36.7 | 34.8 | 33.8 |
| Queens | 61.2 | 59.6 | 63.1 | 66.8 | 63.7 | 65.6 |
| Bronx | 69.5 | 81.6 | 84.9 | 82.4 | 97.9 | 86.2 |
| Suspended sulfates | | | | | | |
| Riverhead | 12.0 | 10.4 | 7.7 | 11.1 | 11.3 | 11.5 |
| Queens | 14.3 | 13.8 | 10.9 | 14.8 | 13.5 | 14.0 |
| Bronx | 13.9 | 14.9 | 12.0 | 16.1 | 14.6 | 14.0 |
| Suspended nitrates | | | | | | |
| Riverhead | 1.4 | 1.3 | 1.0 | 2.1 | 3.1 | 2.5 |
| Queens | 1.7 | 1.6 | 2.7 | 4.6 | 5.3 | 4.0 |
| Bronx | 1.6 | 1.9 | 4.3 | 4.8 | 5.0 | 3.7 |

## Table 5.1.A.8. YEARLY POLLUTANT CONCENTRATIONS FOR NEW YORK CITY, 1944-1970 [a]

| | Citywide | | | | Bronx | | | | Queens | | | | Suffolk County (Riverhead) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Year | Dustfall, g/m²/mo | SO₂, µg/m³ | TSP, µg/m³ | SS,[b] µg/m³ | Dustfall, g/m²/mo | SO₂, µg/m³ | TSP, µg/m³ | SS, µg/m³ | Dustfall, g/m²/mo | SO₂, µg/m³ | TSP, µg/m³ | SS, µg/m³ | Dustfall, g/m²/mo | TSP, µg/m³ |
| 1970 | 8.1 | 210 | 104 | 22.2 | 8.0 | 184 | 111 | 22.2 | 6.0 | 157 | 85 | 5.4 | 2.3 | 39 |
| 1969 | 9.1 | 210 | 98 | 18.2 | 10.0 | 236 | 97 | 10.2 | 6.0 | 131 | 75 | 11.5 | 3.1 | 31 |
| 1968 | 9.0 | 262 | 96 | 21.2 | (11.0) | (320) | (117) | (12.1) | ( 8.0) | (233) | ( 92) | ( 8.8) | | 52 |
| 1967 | (12.0) | 341 | (138) | 32.2 | (12.0) | 394 | (138) | (13.2) | ( 9.7) | 341 | (107) | (10.7) | 1.8 | 35 |
| 1966 | (16.0) | 393 | (176) | 18.7 | (14.0) | 472 | (154) | (15.4) | (12.8) | 420 | (128) | (14.1) | | |
| 1965 | (18.0) | 445 | (190) | 28.8 | (14.0) | 446 | (150) | (15.4) | (14.2) | 446 | (151) | (15.6) | | |
| 1964 | 21.0 | 629 | 222 | 33.1 | (15.5) | (466) | (164) | (17.1) | (16.2) | (484) | (171) | (17.8) | | |
| 1963 | 21.7 | 524 | 282 | 31.3 | (16.5) | (398) | (214) | (18.1) | (18.0) | (435) | (234) | (19.8) | | |
| 1962 | 25.2 | 498 | 250 | 25.3 | (17.4) | (344) | (173) | (19.1) | (20.2) | (398) | (200) | (22.2) | | |
| 1961 | 23.8 | 419 | 267 | (25.0) | (18.6) | (327) | (208) | (20.4) | (22.1) | (390) | (248) | (24.3) | | |
| 1960 | 23.9 | 472 | 230 | (24.0) | 19.9 | (377) | (184) | (21.9) | 24.5 | (472) | (230) | (26.9) | | |
| 1959 | 24.7 | 445 | 180 | (23.0) | 23.2 | (405) | (164) | (25.5) | 23.8 | (418) | (169) | (26.1) | | |
| 1958 | 25.2 | 472 | 165 | 22.8 | 20.1 | (368) | (129) | (22.1) | 24.0 | (444) | (155) | (26.2) | | |
| 1957 | 23.7 | (423) | (188) | 32.1 | 20.5 | (369) | (164) | (22.5) | 22.3 | (401) | (178) | (24.5) | | |
| 1956 | 22.6 | (352) | (155) | 24.2 | 17.2 | (292) | (129) | (18.9) | 20.3 | (345) | (152) | (22.3) | | |
| 1955 | 22.3 | | | 25.6 | 15.5 | | | | 19.7 | | | | | |
| 1954 | 21.6 | | | 22.8 | 15.2 | | | | 19.6 | | | | | |
| 1953 | 23.6 | | | | 16.2 | | | | 23.2 | | | | | |
| 1952 | 20.6 | | | | 14.7 | | | | 19.0 | | | | | |
| 1951 | 25.0 | | | | 17.4 | | | | 25.2 | | | | | |
| 1950 | 28.2 | | | | | | | | | | | | | |
| 1949 | 28.7 | | | | | | | | | | | | | |
| 1948 | 39.2 | | | | | | | | | | | | | |
| 1947 | 41.3 | | | | | | | | | | | | | |
| 1946 | 39.3 | | | | | | | | | | | | | |
| 1945 | 50.7 | | | | | | | | | | | | | |
| 1944 | 56.7 | | | | | | | | | | | | | |

[a] Estimated values are in parentheses.

[b] Data from Manhattan 121st Street station used for citywide suspended sulfate values.

# 5.2 PREVALENCE OF CHRONIC RESPIRATORY DISEASE SYMPTOMS IN ADULTS: 1970 SURVEY OF NEW YORK COMMUNITIES

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## INTRODUCTION

A number of studies have documented the adverse health effects of air pollution on people with established chronic lung disease. Mortality from bronchitis has been observed to increase disproportionately during air pollution episodes.[1-10] Morbidity of people with chronic lung disease was also increased during air pollution episodes, as reflected by the number of hospital emergency clinic visits that occurred during two episodes in New York City.[11,12] Furthermore, prospective studies of large groups of bronchitic subjects have shown a positive correlation between the day-to-day variation in acute symptoms and air pollution levels.[13-15]

The place of long-term air pollution exposure in the etiology of chronic lung disease is harder to define. In studies of acute symptoms, one can usually record factors influencing the incidence of illness, such as smoking habits, occupational exposure to toxic substances, age, place of residence, air pollution exposure, and socioeconomic status. Such factors usually are constant for the duration of the study. However, chronic disease investigations must deal with fluctuation of these important covariates over time. Prospective studies that obtain detailed accurate covariate information have been prohibitively expensive, while retrospective studies require an accuracy of recall over many years that is difficult to attain. Therefore, most chronic disease studies have been cross-sectional comparisons of symptom rates in different areas at the same time.[16-21] These studies have usually assumed that air pollution exposures, smoking habits, socioeconomic status, and place of residence of subjects at the time of study are fair approximations of these characteristics over a lifetime. It is also not possible to find communities alike in every respect except exposure to air pollution. Thus, the evidence adduced from any single cross-sectional study is relatively weak. If the apparent associations shown between air pollution exposure and chronic lung disease are to be considered credible, they must be found at different times by several investigators in different areas.

In the present study, three middle-class communities were chosen to represent a gradient of air pollution exposure. These communities are part of the Environmental Protection Agency's Community Health and Environmental Surveillance System (CHESS) program. Parents of elementary school children in these communities were asked to provide information about their own respiratory symptoms. The study had three purposes: (1) to determine whether chronic respiratory disease symptom rates in adult populations, age 20 to 50, corresponded to a gradient for past or present exposure to air pollution; (2) to initiate a prospective study to determine the effect of pollution control on chronic respiratory disease; and (3) to relate chronic respiratory symptom rates among migrants to their past and recent air pollution exposures.

## METHODS

### Community Selection

Available air quality data for the 25 years preceding the study (1944-1970) were obtained from the New York City Department of Air Resources (NYC-DAR) and the Suffolk County (New York) Department of Health. These data, in conjunction with the most recent available census data and current information on house and property values, were used to select communities alike socioeconomically but different in air pollution exposure.

After considering long-term air pollution exposure trends, Riverhead, Long Island, was chosen as a Low exposure community, the Howard Beach section of Queens as an Intermediate exposure community, and the Westchester section of the Bronx as a High exposure community. As a result of recent improvements in air quality, the Queens and Bronx communities were found to have similar current pollutant concentrations, often below National Primary Air Quality Standards. Hence, on the basis of recent measurements, the latter two communities were redesignated Intermediate I and Intermediate II, respectively. On a historical basis, however, the Intermediate communities have experienced pollution exposures well above those experienced by the Low community.

### Collection of Health and Demographic Data

Parents of all children attending elementary schools located within 1.5 miles of air monitoring stations in the three communities were asked to participate in the study. In May 1970, each child was given a questionnaire at school to be filled out by his parents and returned to school. The questionnaire, based on the standard British Medical Research Council questionnaire on chronic bronchitis, elicited information concerning cough, production of phlegm from the chest, shortness of breath, as well as age, sex, smoking habits, occupational exposure to toxic dust or fumes, and residential mobility. (An example of the form used has been presented elsewhere.)[22] Data on race were obtained from school records. A random subsample of families, stratified by race, was interviewed at home to obtain information on education and income. This subsample contained only whites from the Intermediate I and II communities; both blacks and whites were interviewed in the Low exposure community.

### Assessing Air Pollution

NYC-DAR has maintained monitoring stations within the Intermediate communities for many years. Levels of sulfur dioxide (peroxide titration method), total suspended particulate (24-hour samples), soiling index (coefficient of haze, 2-hour spots), and monthly dustfall were recorded. This information was used to reconstruct past exposures to air pollutants.[23] More recent air quality data collected by the Suffolk County Department of Health confirmed that exposures to air pollutants in the Low exposure community were below the National Primary Ambient Air Quality Standards for suspended particulates and oxides of sulfur. However, very few aerometric data and no emissions inventories were available in Suffolk County, so no detailed estimates of past exposures could be made for the Low exposure community.

Supplementary air monitoring was carried out by the Environmental Protection Agency at sites that were also utilized by agencies of local governments. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high-volume samplers), suspended sulfates (high-volume samplers), and suspended nitrates (high-volume samplers) were collected daily. Dustfall was determined from monthly samples. Twenty-four-hour nitrogen dioxide measurements were also made; however, the validity of the method used (Jacobs-Hochheiser) has subsequently been questioned due to variable collection efficiencies and interferences.

### Analysis of Data

Reported respiratory symptoms were used to devise three indices of morbidity, which were grouped by smoking status and sex within each community. First, chronic bronchitis was defined in accordance with the British Medical Research Council (cough and phlegm for at least 3 consecutive months of the year), and prevalence rates were compared across areas. Second, all respiratory symptoms were used to construct a severity gradient in the following manner:

1. No symptoms.
2. Cough alone for less than 3 months each year.
3. Phlegm with or without cough for less than 3 months each year.
4. Cough without phlegm for 3 months or more each year.
5. Phlegm without cough for 3 months or more each year.
6. Cough and phlegm for 3 months or more each year.
7. Cough and phlegm for 3 months or more each year and shortness of breath.

Respondents were classified according to their reported symptoms and assigned a score corresponding to their rank on this gradient. These scores were then averaged for all respondents to permit area comparisons. The mean respiratory symptom score thus utilized a greater range of symptom information furnished by questionnaire respondents than did the chronic bronchitis prevalence rate. Finally, since the mean respiratory symptom scores are markedly influenced by the number of persons reporting no symptoms, mean severity scores were obtained by averaging symptom scores only for those persons reporting respiratory symptoms. Severity scores are an index of severity, given the presence of symptoms. Bronchitis prevalence rates, symptom scores, and severity scores were adjusted for the covariables of smoking, age, and sex using multivariate techniques. Statistical significance of differences attributable to community exposure was then tested by a linear Chi Square model.[24]

## RESULTS

### Environmental Exposure

New York City pollutant exposures were averaged over periods that roughly reflect major changes in fossil fuel usage (Table 5.2.1). Prior to 1949, the major fuel employed was coal. Unfortunately, reliable aerometric data from this period are too sparse to permit meaningful estimation of sulfur oxide exposures. During the decades 1949-1958 and 1959-1968, petroleum fuels were predominant. These fuels often had a high sulfur content but caused less particulate pollution than coal. In the most recent period, 1969-1971, much emphasis had been placed on control of particulate pollutants and the use of low-sulfur fuels. Estimates of total suspended particulates and sulfur dioxide for the years 1949-1958 were projected from citywide and borough-specific dustfall measurements. Citywide total suspended particulate and sulfur dioxide measurements were available from 1958 and these were used to project borough-wide estimates for Queens and Bronx. These estimates were calibrated using borough-specific monitoring data available for both suspended particulates and sulfur dioxide since 1968. More recent (1971) monitoring data for all study communities are presented in Table 5.2.2.

Two air quality trends are unmistakable. First, the Intermediate communities were exposed to markedly elevated levels of air pollutants for at least 20 years prior to 1970. Second, striking improvements in air quality, as indexed by particulate and sulfur dioxide measurements, were made prior to 1969. From 1949 to 1968, annual average sulfur dioxide levels in the Intermediate communities (estimated at 343 to 404 $\mu g/m^3$) were apparently 4 to 5 times the present National Annual Primary Ambient Air Quality Standard of 80 $\mu g/m^3$, which is based upon the need to protect human health. Moreover, annual suspended particulate concentrations in the Intermediate communities during the same period (estimated at 141 to 173 $\mu g/m^3$) were up to two and one

**Table 5.2.1. ARITHMETIC MEAN POLLUTION CONCENTRATIONS IN NEW YORK CITY, 1949-1970[a]**

| Pollutant and community[b] | Pollutant levels | | |
|---|---|---|---|
| | 1949-58[c] | 1959-68 | 1969-70 |
| Sulfur dioxide, $\mu g/m^3$ | | | |
| Citywide | 416 | 443 | 210 |
| Queens | 397 | 404 | 144 |
| Bronx | 343 | 395 | 210 |
| Total suspended particulate, $\mu g/m^3$ | | | |
| Citywide | 169 | 203 | 101 |
| Queens | 162 | 173 | 80 |
| Bronx | 141 | 166 | 104 |
| Suspended sulfate, $\mu g/m^3$ | | | |
| Citywide | 26 | 26 | 20 |
| Queens | 24 | 19 | 9 |
| Bronx | 22 | 18 | 16 |
| Dustfall, $g/m^2/mo$ | | | |
| Citywide | 24 | 19 | 9 |
| Queens | 22 | 17 | 6 |
| Bronx | 17 | 16 | 9 |

[a] All measurements provided by New York City Department of Air Resources.

[b] Queens and Bronx are the boroughs in which the respective Intermediate I and II communities are located.

[c] Sulfur dioxide and total suspended particulate levels based upon projection from dustfall measurement.

**Table 5.2.2. MEAN ANNUAL POLLUTION CONCENTRATIONS, 1971, NEW YORK CHESS COMMUNITIES**

| Pollutant | Community | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| Sulfur dioxide, $\mu g/m^3$ | | | |
| CHESS | 23 | 51 | 51 |
| NYC-DAR | – | 63 | 58 |
| Total suspended particulate, $\mu g/m^3$ | | | |
| CHESS | 34 | 63 | 86 |
| NYC-DAR | – | 84 | 104 |
| Suspended sulfates, $\mu g/m^3$ | 10.2 | 13.2 | 14.3 |
| Suspended nitrates, $\mu g/m^3$ | 1.9 | 3.5 | 4.1 |
| Dustfall, $g/m^2/mo$ | 1.22 | 3.80 | 5.13 |

half times the National Annual Primary Ambient Air Quality Standard (75 $\mu g/m^3$ geometric mean). High annual sulfate levels (estimated at 18 to 24 $\mu g/m^3$) evidently prevailed as well. Because of stringent control measures, levels of these pollutants have fallen during the last few years. By 1971, suspended particulate levels, though still somewhat above national standards, were only half those encountered in earlier decades. Sulfur dioxide levels, which had been reduced by a factor of 4, were within the recommended standards, and sulfates were reduced to about 14 $\mu g/m^3$. Unfortunately, it was not possible to construct comparable long-term exposure trends for other pollutants such as oxides of nitrogen, carbon monoxide, and various particulate fractions. The available data indicated that differences in the air pollution levels for the two Intermediate communities were not marked even though borough-wide estimates for Queens were somewhat higher than those of the Bronx before 1968 and lower thereafter. Air monitoring data also indicated that Riverhead was indeed a clean community with all pollution levels substantially below those specified by National Ambient Air Quality Standards. Suspended particulate sulfate levels in Riverhead were somewhat elevated, perhaps reflecting the intrusion of fine particulate from more central areas of the New York-New Jersey conurbation.

**Table 5.2.3. NUMBER AND PERCENT OF FAMILIES RETURNING QUESTIONNAIRE BY COMMUNITY**

| Community | Families returning questionnaire | Percent response |
|---|---|---|
| Low | 1367 | 73 |
| Intermediate I | 1529 | 78 |
| Intermediate II | 1868 | 85 |
| Total | 4764 | 78.4 |

## Response Rates

Questionnaires were completed and returned to the schools by 78 percent of the 4764 families to whom they were sent (Table 5.2.3). Response rates were somewhat better in the Intermediate areas (78 and 85 percent, respectively, in the Intermediate I and II communities) than in the Low pollution area (73 percent). If white respondents alone were considered, area differences in response rates would be lessened, with the Low and Intermediate I exposure areas becoming almost identical. It was not possible to interview nonrespondents personally.

## Characterization of the Study Population

The study population was predominantly white, although a sizeable black segment resided in the Low pollution community and a small black segment resided in the Intermediate I community. Since the overall size of the black populations proved too small for meaningful analysis of blacks alone, only whites were considered in the analyses. The age distribution of parents (Table 5.2.4) reflects the method of population selection. Overall, about 50 percent of the parents were in the 31- to 40-year-old age group. There were no significant intercommunity differences in age distribution within smoking- and sex-specific categories. Smoking histories were obtained for parents both to determine community smoking patterns and to adjust for this variable in the evaluation of respiratory symptoms. Cigarette smoking patterns were quite similar for the three study populations: between 40 and 45 percent of each population were smokers, and 35 to 40 percent of these smoked at least 15 cigarettes daily. Although the proportion of smokers was approximately equal in women and men, men more often smoked one or more packs of cigarettes per day. In this study, about 20 percent of the total population were exsmokers, and these were classified separately for most analyses; however, sample size considerations necessitated pooling current smokers and exsmokers for the analysis of migration effects. Since the Low pollution community had a somewhat greater proportion of exsmokers (25 percent compared with 20 and 16 percent), such pooling would probably tend to reduce symptom rates and symptom severity for that community.

**Table 5.2.4. DISTRIBUTION OF STUDY POPULATION BY COMMUNITY, SMOKING STATUS, SEX, AND AGE[a]**

| Category | Community | | | Total |
|---|---|---|---|---|
| | Low | Intermediate I | Intermediate II | |
| Nonsmokers | | | | |
| Mothers | | | | |
| ≤40 | 148 | 298 | 380 | 826 |
| >40 | 49 | 113 | 149 | 311 |
| Fathers | | | | |
| ≤40 | 104 | 229 | 288 | 621 |
| >40 | 70 | 155 | 211 | 436 |
| Exsmokers | | | | |
| Mothers | | | | |
| ≤40 | 123 | 190 | 177 | 490 |
| >40 | 21 | 43 | 49 | 113 |
| Fathers | | | | |
| ≤40 | 99 | 142 | 123 | 364 |
| >40 | 45 | 80 | 75 | 200 |
| Smokers | | | | |
| Mothers | | | | |
| ≤40 | 207 | 449 | 481 | 1137 |
| >40 | 60 | 86 | 126 | 272 |
| Fathers | | | | |
| ≤40 | 139 | 327 | 338 | 804 |
| >40 | 77 | 165 | 188 | 430 |
| Total | 1142 | 2277 | 2585 | 6004 |

[a] Only whites were utilized because small black population samples precluded meaningful analyses.

Comparison of socioeconomic patterns (Table 5.2.5) indicated that socioeconomic differences did exist, with the Intermediate I community ranking above the Low and Intermediate II communities. The Intermediate I community was the best educated, had the highest family income, and was the least crowded in living accommodations. The Intermediate II community had the poorest educational background, showed the lowest family income, and was the most crowded in housing. The Low pollution community ranked intermediate for all three variables.

Less than 1 percent of the population in any of the study communities reported occupational exposure to irritating smoke, dust, or fumes; therefore, no separate analysis of these individuals was performed.

## Chronic Respiratory Disease Evaluation

### *Chronic Bronchitis Prevalence*

Prevalence rates for chronic bronchitis were computed for the combined Intermediate and Low exposure populations by age, sex, and

**Table 5.2.5. COMMUNITY CHARACTERISTICS: FAMILY INCOME, RACIAL COMPOSITION, AND EDUCATIONAL STATUS OF FATHERS (percent)**

| Characteristic | Community | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| White[a] | 64.6 | 93.4 | 88.0 |
| High school graduate[b] | 63.0<br>(246) | 49.1<br>(281) | 55.3<br>(235) |
| College graduate[b] | 13.4<br>(246) | 24.2<br>(281) | 7.2<br>(235) |
| Family income ≤$8000[b] | 23.1<br>(238) | 8.7<br>(276) | 33.9<br>(224) |

[a] Based on entire population returning questionnaires.
[b] Based on subsample of whites interviewed in their homes. Numbers in parentheses represent size of subsample.

smoking habit to determine the overall effects of these variables (Table 5.2.6). As expected, rates in all age-sex categories were higher in smokers than nonsmokers. Chronic bronchitis rates for mothers who were exsmokers closely approximated rates for nonsmokers; fathers who were exsmokers reported rates intermediate between those reported for lifetime nonsmokers and those found in current cigarette smokers. Bronchitis rates were also higher in fathers than in mothers in all of the age- and smoking-specific comparisons. No consistent upward trend in bronchitis prevalence was noted with aging in this relatively young population.

On the other hand, comparison of smoking- and sex-specific rates between the Intermediate and Low communities showed marked area differences for smokers, exsmokers, and nonsmokers of both sexes (Table 5.2.7). For nonsmokers, bronchitis rates in the combined Intermediate communities were 2 to 4 times those in the Low pollution community. Although this relative difference was less striking in exsmokers and smokers, the absolute differences in rates were generally similar. Much smaller, inconsistent differences were observed between the two Intermediate communities. In each community, mothers–whether smokers, exsmokers, or nonsmokers–had lower rates than fathers. Tests for statistical significance of these effects as well as the effect of age are presented separately for mothers and fathers in Table 5.2.8. Differences in rates between the three communities and between the Low and combined Intermediate communities were significant for both mothers and fathers ($p < 0.01$). Differences between the two Intermediate communities were not significant. No significant age effect was seen in either mothers or fathers; but as expected, smoking was consistently highly significant. Among mothers, there were no significant differences between lifetime nonsmokers and exsmokers; however, significant differences were found among fathers.

*Mean Respiratory Symptom and Severity Scores*

Respiratory symptom and severity scores were also summarized for the three communities by smoking status for mothers and for fathers. The mean symptom score is the average symptom rating in the total subgroup designated; the mean severity score represents the average symptom rating in only those who reported symptoms.

Mean respiratory symptom scores (Table 5.2.9) for residents of the Intermediate communities, like chronic bronchitis prevalence rates, were consistently higher than scores for the Low exposure community.

**Table 5.2.6. AGE-, SMOKING-, AND SEX-SPECIFIC CHRONIC BRONCHITIS PREVALENCE RATES[a]**
**(percent)**

| Category | Age ≤40 | Age >40 |
|---|---|---|
| **Nonsmokers** | | |
| Mothers | 5.7 | 4.5 |
| Fathers | 12.9 | 15.6 |
| **Exsmokers** | | |
| Mothers | 6.9 | 4.4 |
| Fathers | 17.3 | 17.0 |
| **Smokers** | | |
| Mothers | 17.0 | 18.8 |
| Fathers | 20.1 | 20.7 |
| **Overall rate** | 13.6 | 14.8 |
| **Sample size** | 4242 | 1762 |

[a] Crude rates for symptom severity 6-7.

**Table 5.2.7. SMOKING- AND SEX-SPECIFIC CHRONIC BRONCHITIS PREVALENCE RATES DISTRIBUTED BY COMMUNITY**
**(percent)**

| Category | Community: Low | Intermediate I | Intermediate II |
|---|---|---|---|
| **Nonsmokers** | | | |
| Mothers | 2.0 | 7.5 | 4.9 |
| Fathers | 4.6 | 18.0 | 14.2 |
| **Exsmokers** | | | |
| Mothers | 3.8 | 9.0 | 4.5 |
| Fathers | 13.9 | 18.0 | 18,7 |
| **Smokers** | | | |
| Mothers | 13.9 | 19.8 | 16.6 |
| Fathers | 13.9 | 21.3 | 22.1 |
| **Smoking-adjusted rates** | | | |
| Mothers | 4.7 | 11.6 | 10.6 |
| Fathers | 11.5 | 18.4 | 17.4 |

**Table 5.2.8. ANALYSES OF VARIANCE FOR HEALTH OBSERVATIONS, CHRONIC BRONCHITIS PREVALENCE RATES**

| Factor | Degrees of freedom | Mothers | | Fathers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability(p) | $X^2$ | Probability(p) |
| Community prevalence rates | 2 | 9.48 | 0.0091 | 28.07 | 0.0001 |
| Low vs pooled Intermediate | 1 | 7.84 | 0.0054 | 28.07 | 0.0001 |
| Intermediate I vs Intermediate II | 1 | 2.14 | 0.14 | 0.00 | >0.5 |
| Smoking | 2 | 73.72 | 0.0001 | 20.18 | 0.0002 |
| Exsmokers vs nonsmokers | 1 | 0.43 | >0.5 | 5.37 | 0.02 |
| Age | 1 | 0.11 | >0.5 | 0.00 | >0.5 |
| Model fit | 4 | 1.08 | 0.90 | 1.85 | 0.77 |

Moreover, smoking-adjusted differences between the two Intermediate communities faithfully mirrored the previously described differences in chronic bronchitis prevalence. In both sexes, scores for exsmokers were intermediate between the higher scores of current smokers and the lower scores of lifetime nonsmokers. An analysis of variance was performed, testing the effects of community pollution exposure, of smoking, and of age upon mean symptom score (Table 5.2.10). The results mimicked those of the analysis testing the effects of these variables on chronic bronchitis prevalence rates. Thus, mean symptom scores proved a useful confirmatory analysis.

**Table 5.2.9. SMOKING- AND SEX-SPECIFIC MEAN RESPIRATORY SYMPTOM SCORES DISTRIBUTED BY COMMUNITY**
**(percent)**

| Category | Community | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| **Nonsmokers** | | | |
| Mothers | 1.29 | 1.76 | 1.61 |
| Fathers | 1.81 | 2.41 | 2.35 |
| **Exsmokers** | | | |
| Mothers | 1.37 | 2.00 | 1.72 |
| Fathers | 2.05 | 2.56 | 2.51 |
| **Smokers** | | | |
| Mothers | 2.30 | 2.68 | 2.53 |
| Fathers | 2.48 | 2.76 | 2.76 |
| **Smoking-adjusted rates** | | | |
| Mothers | 1.61 | 2.07 | 2.02 |
| Fathers | 2.11 | 2.57 | 2.52 |

Sex- and smoking-specific mean severity scores were then calculated (Table 5.2.11) and statistically tested by the analysis of variance model previously described (Table 5.2.12). These conditional severity scores were consistently and significantly higher for males, both smokers and nonsmokers, living in the Intermediate communities. Severity scores were generally higher for male exsmokers than for males who still smoked cigarettes. This result suggested that the presence of respiratory symptoms may have been related to cessation of smoking, with the more symptomatic males being more likely to stop smoking. Severity scores for female exsmokers were intermediate between scores for lifetime nonsmokers and for current cigarette smokers. This result suggested that factors other than the threat of current respiratory symptoms induced mothers of young children to stop smoking cigarettes.

**Table 5.2.10. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS, MEAN RESPIRATORY SYMPTOM SCORES**

| Factor | Degrees of freedom | Mothers | | Fathers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability(p) | $X^2$ | Probability(p) |
| Community symptom scores | 2 | 25.32 | 0.0001 | 25.27 | 0.0001 |
| Low vs pooled Intermediate | 1 | 23.95 | 0.0001 | 25.73 | 0.0001 |
| Intermediate I vs Intermediate II | 1 | 2.11 | 0.14 | 0.02 | >0.5 |
| Smoking | 2 | 144.37 | 0.0001 | 29.14 | 0.0001 |
| Exsmokers vs nonsmokers | 1 | 2.36 | 0.12 | 2.52 | 0.11 |
| Age | 1 | 0.23 | >0.5 | 0.06 | >0.5 |
| Model fit | 4 | 1.79 | 0.78 | 5.33 | 0.255 |

### *Effects of Migration on Prevalence of Chronic Bronchitis*

Based upon questionnaire responses, the study population was grouped into four categories: those who had always lived in New York City; those who moved to New York City from relatively clean, smaller cities of less than 200,000 population; those who moved into the Low exposure community from a city larger than 200,000; and those who had always lived in relatively clean areas. Respondents who reported more than one move across the clean and polluted areas were excluded from the analyses. Because the sample of exsmokers was small, this group was combined with current smokers in the data analysis. The proportions of males and females were similar in each residence-smoking category. Chronic bronchitis prevalence rates for each residence-smoking category (Table 5.2.13) were calculated and found to be 1.6 to 5 times higher in smokers than in lifetime nonsmokers. A novel and potentially important finding was that migrants to polluted areas had developed elevated bronchitis rates similar to those of lifelong residents of the polluted communities. Migrants to the Low exposure community exhibited chronic bronchitis prevalence rates that were similar to those of lifelong residents of relatively clean areas. Unfortunately, sample size limitations precluded estimating a time interval of exposure to which one could attribute either beneficial or harmful effects.

### *Effect of Air Pollution Relative to Smoking*

To assess the relative importance of ambient air pollution and cigarette smoking, smoking- and area-specific prevalence rates were compared

**Table 5.2.11. MEAN SEVERITY SCORES IN PRESENCE OF ANY CHRONIC RESPIRATORY DISEASE SYMPTOM**

| Category | Community | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| Nonsmokers | | | |
| Mothers | 4.40 | 4.45 | 4.37 |
| Fathers | 4.19 | 4.88 | 4.68 |
| Exsmokers | | | |
| Mothers | 4.58 | 4.67 | 4.62 |
| Fathers | 4.77 | 4.79 | 4.99 |
| Smokers | | | |
| Mothers | 4.67 | 4.86 | 4.69 |
| Fathers | 4.56 | 4.73 | 4.82 |
| Smoking-adjusted rates | | | |
| Mothers | 4.44 | 4.72 | 4.88 |
| Fathers | 4.57 | 4.85 | 5.01 |

Table 5.2.12. ANALYSIS OF VARIANCE FOR HEALTH OBSERVATIONS, SEVERITY SCORES FOR THOSE WITH SYMPTOMS

| Factor | Degrees of freedom | Mothers | | Fathers | |
|---|---|---|---|---|---|
| | | $X^2$ | Probability(p) | $X^2$ | Probability(p) |
| Community severity scores | 2 | 0.81 | >0.5 | 6.62 | 0.04 |
| Low vs pooled Intermediate | 1 | 0.00 | >0.5 | 6.62 | 0.01 |
| Intermediate I vs Intermediate II | 1 | 0.81 | >0.5 | 0.00 | >0.5 |
| Smoking | 2 | 2.94 | 0.23 | 4.64 | 0.10 |
| Exsmokers vs nonsmokers | 1 | 2.33 | 0.12 | 4.64 | 0.03 |
| Age | 1 | 0.45 | >0.5 | 0.40 | >0.5 |
| Model fit | 4 | 5.65 | 0.23 | 0.26 | >0.5 |

Table 5.2.13. ASSOCIATION OF CHANGES IN CHRONIC BRONCHITIS PREVALENCE WITH CHANGES IN POLLUTION EXPOSURE[a]

| Exposure history | Chronic bronchitis prevalence rate, % | | | |
|---|---|---|---|---|
| | Nonsmokers | | Exsmokers and current cigarette smokers | |
| | Rate | Population at risk | Rate | Population at risk |
| Always lived in New York City | 9.2 | 1000 | 19.3 | 2743 |
| Moved to New York City from less populous area | 10.8 | 139 | 15.8 | 152 |
| Moved to Low exposure area from large city | 2.0 | 100 | 10.1 | 298 |
| Always lived in relatively clean area | 2.6 | 192 | 12.3 | 527 |

[a] Both sexes combined.

(Table 5.2.14). Rates for mothers and fathers were compared separately. In each case, the base rate was the prevalence of chronic bronchitis among lifetime nonsmokers living in the Low community. The experience of the two Intermediate communities was pooled. The excess prevalence attributable to smoking was computed by subtracting the chronic bronchitis prevalence among nonsmoking fathers (or mothers) in the Low community from the prevalence among smoking fathers (or mothers) in the Low community. The excess prevalence attributable to pollution was computed by subtracting the chronic bronchitis prevalence among nonsmokers in the Low community from the prevalence among nonsmokers in the Intermediate communities. The excess prevalences attributable to smoking and to pollution among exsmokers were computed in the same manner, using prevalence rates of exsmokers instead of current smokers.

One measure of the importance of ambient air pollution relative to smoking may be expressed by a ratio whose numerator is the excess prevalence attributable to pollution and whose denominator is the excess prevalence attributable to present or past smoking. When such ratios were computed for females, air pollution was found to have 34 percent the effect of current cigarette smoking and 222 percent the effect of former cigarette smoking. For males, ambient air pollution was computed to have 122 percent the effect of either current or former cigarette smoking. Overall, air pollution seemed almost (78 percent) as important a factor as current cigarette smoking and more important (172 percent) than previous cigarette smoking.

If the hazards of self-pollution by cigarette smoking and ambient air pollution were additive, then the sum of the excess prevalence for smokers living in the Low community and the excess prevalence for nonsmokers from the Intermediate communities should equal the excess prevalence for smokers living in Intermediate communities. The same procedure may be applied for exsmokers. When these procedures were applied, the combination of current cigarette smoking and air pollution was found to explain 83 to 101 percent of the expected excess bronchitis under a simple additive model. The comparable values when the additive model involved exsmokers were 67 to 83 percent. Together, these models suggested that the effects

**Table 5.2.14. RELATIVE IMPORTANCE OF CIGARETTE SMOKING AND AMBIENT AIR QUALITY AS DETERMINED BY COMPARISON OF EXCESS PREVALENCE OF CHRONIC BRONCHITIS[a]**

| Current cigarette smoking status | Community | Excess prevalence of chronic bronchitis | Simple additive model combining adverse effects of smoking and pollution | |
|---|---|---|---|---|
| | | | Expected | Observed/expected |
| Female | | | | |
| Lifetime nonsmoker | Low | 0.00(2.0) | — | — |
| | Intermediate | 4.0 | — | — |
| Exsmoker | Low | 1.8 | — | — |
| | Intermediate | 4.8 | 5.8 | 0.83 |
| Smoker | Low | 11.9 | — | — |
| | Intermediate | 16.1 | 15.9 | 1.01 |
| Male | | | | |
| Lifetime nonsmoker | Low | 0.00(4.6) | — | — |
| | Intermediate | 11.3 | — | — |
| Exsmoker | Low | 9.3 | — | — |
| | Intermediate | 13.7 | 20.6 | 0.67 |
| Smoker | Low | 9.3 | — | — |
| | Intermediate | 17.1 | 20.6 | 0.83 |

[a] Base rates in parentheses. Excess prevalence = smoking-and area-specific prevalence rate minus base rate.

of ambient air pollution and smoking were roughly additive.

## DISCUSSION

In this study, chronic respiratory disease frequency, whether defined by symptom score or as a symptom profile compatible with chronic bronchitis, was significantly higher in the two Intermediate communities. People living in the Low pollution community had a markedly lower prevalence of respiratory symptoms than people living in the Intermediate communities. This difference was observed for people over as well as under 40 years of age; for both sexes; and for smokers, exsmokers, and nonsmokers.

Two important additional observations strengthen the association of ambient air pollution with chronic respiratory disease symptoms: First, migrants moving to the Low community from polluted communities had lower rates than lifetime residents of the Intermediate communities, while migrants to the Intermediate communities from clean communities were found to assume the higher rates of the Intermediate communities. Second, excess prevalence models indicated that the hazards of air pollution exposure and continued or past self-pollution by cigarette smoking were nearly additive. These models also indicated that under the circumstances of this particular study, the contribution of air pollution to observed prevalence rates, relative to smoking, was substantial. The figures describing this contribution are not meant to be exact measurements; rather, they are intended to point out that pollution may exert a stronger effect than had been previously suggested.

The observed effects of covariates in the present study were consistent with previous findings on the determinants of chronic bronchitis in that smokers and males were found to have substantially higher rates than nonsmokers and females. Failure to define a linear age gradient may be explained in part by the relatively young age of the study population.

Before assessing the full implications of the present report, critical consideration must be given to the impact of four factors: socioeconomic differences, ethnic exclusion, nonrespondent morbidity, and differences between recent and remote air pollution exposures.

First, if one considers that lower socioeconomic status might be associated with an increase in chronic bronchitis prevalence rates, then the documented intercommunity socioeconomic differences would dampen the observed morbidity excess found when the more prosperous Intermediate I community was compared to the less advantaged Low pollution area. Conversely, the same socioeconomic effect would accentuate observed morbidity differences between the less prosperous Intermediate II community and the relatively more prosperous Low pollution area. For this particular study, the opposite argument, that higher socioeconomic status might be associated with an increase in reported prevalence rates, might be applied in view of the fact that the more prosperous Intermediate I community reported the highest rates. In either case, socioeconomic bias in this study would be minimal because of the intermediate socioeconomic status of the Low pollution community. It is extremely unlikely that any residual socioeconomic bias could explain the substantial intercommunity prevalence differences that were observed.

Second, ethnic exclusions facilitated the analyses, but inclusion of blacks would not have altered the findings of the study. Blacks and whites exhibited the same symptom gradient between Low and Intermediate communities, and there were no significant differences attributable to racial differences alone. The only anomalous finding was that black male nonsmokers had somewhat higher chronic bronchitis prevalence and mean symptom scores than black male smokers. This anomaly could well be explained by the relatively small sample size of blacks.

Third, morbidity in nonrespondents might dilute the observed differences in morbidity. For this to occur, area differences in morbidity reporting among nonrespondents would have to be opposite to the demonstrated trend in morbidity. To equalize the observed area differences, chronic bronchitis symptoms among the nonrespondents in the Low area would have to average over 4 times the rates reported for respondents. In addition, the assumption depends upon none of the nonrespondents in the Intermediate communities having chronic bronchitis. Such a set of events would be indeed extraordinary.

Fourth, estimating past and present ambient and indoor air pollution exposures is a difficult problem that has not been satisfactorily resolved. Nevertheless, particulate and sulfur dioxide pollution in past decades was estimated. More precise measurements were made for current exposures, and the apparent beneficial and detrimental influences of migration and nonmigration were assessed. It was not possible to differentiate or quantify the importance of repeated short-term peak exposures relative to longer lasting low-level pollutant exposures. Personal pollution by cigarette smoking has been duly considered in this report. The importance of indoor air pollutants, such as nitrogen oxides from gas appliances and smoke from other people's cigarettes, has yet to be determined.

There is reasonable evidence that residents of the Intermediate communities were exposed to very high pollution levels during their childhood and/or young adult years. Such early exposures may have contributed to the prevalence of chronic respiratory disease in later life. Symptomatic improvement, however, might eventually be expected if observations based upon the experience of migrants and exsmokers are valid.

Even if childhood exposure to air pollution was not a factor in the development of chronic bronchitis, present pollution levels may still cause such symptoms to persist and perhaps even to worsen. More optimistically, the tide of excess morbidity may already have begun to ebb, and this decrease could accelerate as air quality is improved. On the other hand, one could interpret the present study as suggesting that as yet unspecified pollutants capable of inducing or aggravating chronic respiratory disease are not well controlled. In any event, a single cross-sectional study cannot provide the definitive explanation. A mosaic of information involving multiple epidemiologic and laboratory studies is clearly indicated.

Based upon the available data, we believe it is prudent to consider that long-term exposures to annual average sulfur dioxide levels of about 144 to 404 $\mu g/m^3$ coupled with annual average suspended particulate levels of about 80 to 173 $\mu g/m^3$ and suspended sulfate levels of about 9 to 24 $\mu g/m^3$, in the presence of other fossil fuel combustion products, substantially increases the risk of chronic bronchitis. It is also possible that lower pollution levels may well aggravate or support the persistence of chronic respiratory disease symptoms. Annual sulfur dioxide levels of 50 to 60 $\mu g/m^3$ accompanied by annual average suspended sulfate levels of about 14 $\mu g/m^3$ and annual arithmetic mean total suspended particulate levels of about 60 to 105 $\mu g/m^3$ could be associated with such effects. It is thus quite possible that the present National Primary Ambient Air Quality Standards for sulfur dioxide and suspended particulates contain either a small safety factor or no safety factor at all. Finally, increasing attention should be devoted to elucidating the health implications of suspended particulate sulfates, which are evidently decreasing more slowly than sulfur dioxide or suspended particulates.

## SUMMARY

Chronic respiratory disease morbidity was assessed in 6004 parents of elementary school children residing in either a clean Long Island community or one of two more polluted communities in New York City. Long-term exposures (up to 20 years) to sulfur dioxide levels of an estimated 144 to 404 $\mu g/m^3$ and suspended sulfate levels of an estimated 9 to 24 $\mu g/m^3$ were associated with marked increases in chronic bronchitis prevalence among smokers, exsmokers, and nonsmokers of both sexes age 20 to 50. Shorter term, more recent exposures to lower pollutant levels may also induce, aggravate, or sustain chronic respiratory disease symptoms. Such short-term exposures might involve annual average sulfur dioxide levels of 50 to 60 $\mu g/m^3$ accompanied by annual average suspended sulfate levels of about 14 $\mu g/m^3$ and total suspended particulate levels of about 60 to 105 $\mu g/m^3$. In the present study, the observed differences between communities could not be accounted for by age, occupational exposures, smoking habits, or ethnic factors. It is unlikely that nonrespondent bias or intercommunity socioeconomic differences could have explained the observed intercommunity differences in prevalence rates. An analysis of migration patterns indicated that improvement of air quality was associated with striking decreases in chronic bronchitis prevalence and that moving into a polluted city from a relatively clean area was associated with an increased risk of chronic bronchitis. Air pollution and self-pollution by cigarette smoking appeared to be additive hazards. Improvements in air quality may be expected to benefit the population by reducing chronic respiratory disease symptom frequency and severity.

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# 5.3 PROSPECTIVE SURVEYS OF ACUTE RESPIRATORY DISEASE IN VOLUNTEER FAMILIES: 1970-1971 NEW YORK STUDIES

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## INTRODUCTION

Acute respiratory infections consistently rank as the leading cause of acute illness in the United States. During the influenza year July 1968 to June 1969, the average American endured 1.2 acute respiratory illnesses, which was about twice the expected rate for a noninfluenza year.[1] The direct economic costs of morbidity from acute respiratory infections are difficult to determine precisely but are estimated to be well over $5 billion per year.[2] Children suffer the highest incidence of acute respiratory disease, about twice that of adults, and there is increasing evidence that a history of repeated acute lower respiratory illnesses in childhood may contribute to the development of chronic bronchitis in later life.[3]

Accordingly, one way to reduce the frequency of chronic respiratory disease in adults may be to identify and control factors that might induce or aggravate acute respiratory illnesses in children. Exposure to high levels of atmospheric pollution, as indexed by elevated levels of sulfur dioxide and total suspended particulates, may be an important determinant of acute respiratory illness. British investigators evaluated the effects of long-term exposures among more than 3000 children living in areas representing different levels of air pollution.[4] They found an excess number of lower respiratory tract illnesses in children from the most polluted areas and a significant trend towards more hospitalization for lower respiratory tract infections in the same group of children. A second English study confirmed the association of lower respiratory tract illnesses with elevated pollution levels and implicated air pollution as an important determinant of upper respiratory disorders.[5] In another study, Japanese students living in a highly polluted area reported a higher frequency of respiratory symptoms than did children in a less polluted area.[6]

The laboratory evidence accumulated thus far on the toxicological effect of sulfur dioxide indicates that the pure gas is a mild respiratory irritant that is largely absorbed in the upper respiratory tract. The basic physiologic response appears to be a mild degree of broncho-constriction that causes a measurable increase in flow resistance. However, sulfur dioxide in combination with suspended particulates was found to be more irritating than sulfur dioxide alone.[7,8] Few clinical or toxicological studies involving sulfur dioxide, suspended particulates, or suspended sulfates in combination with various metals have been reported.[9]

The prospective epidemiologic study reported here considered the acute respiratory illness experience of families living in three areas of metropolitan New York. For the period late September 1970 to early May 1971, panels of volunteer families residing in two more urban communities were compared with families living in a less polluted suburban fringe community. An important advantage of the New York study setting was the availability of historic air monitoring data from which estimated past exposures could be reconstructed. The recent rapid decline in air pollutant levels in the area also permitted the investigators to search for early health benefits from air pollution control.

The study tested two hypotheses: first, that the frequency and/or severity of acute respiratory illness would be higher in the more polluted communities; and second, if increases were observed, that at least a portion of the increased vulnerability could be attributed to air pollution exposures of a defined duration. In addition, an attempt was made to quantify the effects of indoor air pollutants and to measure the importance of residential mobility, socioeconomic status, and the history of chronic respiratory disease in parents as determinants of acute respiratory illness in children.

## METHODS

### Setting and Study Population

Three areas that were similar with regard to socioeconomic factors, ethnic composition, and meteorological conditions but differed from each other with respect to atmospheric pollution were selected for study. After considering long-term air pollution exposure trends, Riverhead, Long Island, was chosen as a Low exposure community, the Howard Beach section of Queens as an Intermediate exposure community, and the Westchester section of the Bronx as a High exposure community. As a result of recent improvements in air quality, the Queens and Bronx were found to have similar current pollutant concentrations, often below National Primary Air Quality Standards. Hence, the latter two communities were redesignated Intermediate I and Intermediate II, respectively.

Initial contact with the population residing in a community was made with the cooperation of local school boards, who permitted questionnaires[10] to be distributed to parents by their children who attended specific schools. Information obtained from the questionnaire identified the demographic characteristics of the study population. Comparison of the population returning the completed questionnaire with a sample of the approximately 10 percent who did not indicated that the two groups did not differ demographically.

Panels of subjects selected for study were restricted to families who resided within 1 to 1.5 miles of the air monitoring station, had a functioning telephone, had at least one school child 12 years of age or younger, and had lived at their present address for at least 1 year. Whenever possible, selections were based on the presence of preschool siblings. The study was limited to white families because the paucity of black families living in the more polluted communities precluded ethnic matching. A home visit was made to each prospective study family to gain their willingness to participate and to collect additional information regarding age, occupational pollution, smoking habits, use of gas in the home, and symptoms of chronic respiratory disease. These covariates, with the exception of occupational exposure, were all considered in subsequent analyses. Since the survey indicated that fewer than 1 percent of the fathers worked in a polluted environment, this factor was considered to be insignificant.

## Illness Monitoring

Families were called once every 2 weeks and questioned about the presence of illness, fever, respiratory symptoms, restricted activity, including absences from school or from work or confinement to bed, and whether contact had been made with a physician either by phone or office visit. If a family member had visited a physician, information was elicited concerning the physician's diagnosis. A standardized questionnaire[10] was used to obtain the information and the telephone interviews were conducted by trained interviewers. Whenever possible, information on symptoms was obtained from the mother or female guardian, and determination of the presence or absence of illness was left to her discretion. If the mother was not available, the information was obtained from whomever was considered to be the most responsible resident in the home.

The upper respiratory illness classification was given to those reporting any one of the following: cough (dry, nonproductive), head cold, sore throat, sinus or postnasal drip, or runny nose. Lower respiratory illnesses were characterized by a chest cold, a deep productive chest cough, wheezing, croup, bronchitis, or pneumonia. Whenever both upper and lower tract symptoms were present, the diagnosis of acute lower respiratory illness was arbitrarily assigned. As a check of reliability of reporting, randomly selected households were reinterviewed several days after the routine calls. This reinterview was assigned to experienced interviewers who had not previously contacted the families that were being checked.

## Monitoring Air Pollution Exposures

Measurements of pollutant levels were obtained through the cooperation of the New York City Department of Air Resources (NYC-DAR) and the Suffolk County (New York) Department of Health and from CHESS air monitoring stations located within each community studied. Stations were located on roof tops 30 to 45 feet above ground level. At each station, continuous measurements were obtained for sulfur dioxide (modified West-Gaeke method), total suspended particulates, suspended sulfates, and suspended nitrates. Nitrogen dioxide measurements were made by the Jacobs-Hochheiser method, but because it is now known that this method does not provide accurate data in some instances, the information was not used in the analysis. Newer knowledge related to the inaccuracy of the Jacobs-Hochheiser method indicates that the method overestimates actual levels lower than 90 $\mu g/m^3$. Since levels measured during the period of this study were consistently well below 90 $\mu g/m^3$, it is believed that nitrogen dioxide did not contribute significantly to impairments in health associated with exposure to air pollution. Analyses of air pollution measurements were made on the basis of 24-hour samples. In addition, monthly dustfall samples were obtained. The air monitoring procedures were detailed elsewhere.[11] Historical data were obtained from aerometric stations maintained by NYC-DAR in the Intermediate communities. These stations monitored sulfur dioxide (peroxide titration method), total suspended particulates, monthly dustfall, and soiling index. From these data, estimates of the annual average air pollutant exposures for the decade prior to the study were reconstructed.[11]

### Statistical Analysis

Provisions were made to partition the total study period into four segments so that separate analyses might be focused on any period of epidemic illness. This procedure also minimized the adverse effects of family withdrawals or temporary nonparticipation, since the experience of a family was not considered in the analysis if contact could not be made during any portion of a particular 8-week study segment. Attack rates for upper, lower, and total acute respiratory illness were computed separately for four family segments: fathers, mothers, elementary school children, and preschool children. The experiences of older children or other adults living in the home were excluded from the analyses. The population reporting repeated bouts of acute respiratory illness was defined by considering the illness experience of each volunteer. Intercommunity differences in the frequency of acute respiratory illness were ascertained by comparing two measures: (1) attack rates for acute respiratory illness and (2) the proportion of the study population reporting excess respiratory illness. Repeated excess acute respiratory illness during the study was defined as more than two episodes of either upper or lower tract illness.

Intercommunity differences in illness severity were ascertained by comparison of severity scores, which ranged from Level I through Level V. Briefly, Level I denoted illness without restricted activity, Level II included restricted activity but not fever, Level III included restricted activity and fever, and Level IV included all of the preceding plus a physician visit. Level V applied only to children and was defined as restricted activity and fever accompanied by otitis media (middle ear infection) diagnosed by a physician. Level V was chosen to reflect a common, presumably bacterial, complication of acute upper respiratory illness. Levels II through V were further characterized by the mean number of restricted activity days incurred.

The effects of probable determinants of illness were assessed by comparison of specified or adjusted attack rates and by statistical tests using a general linear model for categorical data.[12]

## RESULTS

### Air Pollution Exposures

Respiratory defense mechanisms might be impaired by either short-term pollutant exposures, long-term exposures, or both. The present study considered both types of exposure. Long-term exposures were indexed by annual average pollutant levels during the study and by estimates for the prior decade, which coincided with early growth and development years of the childhood population. Short-term air pollutant exposures were indexed by the median, ninetieth percentile, and maximum levels during the 32-week study period.

As expected, the Low exposure community had lower pollutant levels than the two Intermediate exposure areas during the study, and evidence indicated that these conditions had existed during the previous decade as well (Table 5.3.1). Suspended sulfate levels, however, were higher than expected in the Low exposure area even though no large point emission sources of sulfur oxides were nearby. The two Intermediate exposure areas generally had higher pollutant levels than the Low exposure area and were similar to each other. During the study, the Intermediate communities had annual average sulfur dioxide levels that approximated the annual average specified by the National Secondary Air Quality Standard (60 $\mu g/m^3$ or 0.02 ppm). Total suspended particulate levels approximated or just exceeded the annual geometric average level specified by the National Primary Air Quality Standard (75 $\mu g/m^3$). Suspended particulate sulfate levels (13 to 14 $\mu g/m^3$) were elevated in both of the Intermediate communities compared to the Low exposure community. Suspended particulate nitrates were similar in the two Intermediate communities ($\sim$4 $\mu g/m^3$) and were also higher than in the Low community. The Intermediate I community was adjacent to a large international airport and may have been polluted with higher levels of hydrocarbons or other materials associated directly with this facility. Because the full potential impact of these pollutants was not recognized at the time the study was initiated, the siting of the study community was considered to be acceptable, and aerometric measurements of hydrocarbons were not included in the study design.

Air pollution levels during the decade prior to the study were indicated to be much higher than present values in both of the Intermediate exposure communities. Sulfur dioxide concentrations were probably 3 to 5 times the current Primary Air Quality Standard, and suspended particulate concentrations were approximately twice the current standard. Estimated suspended sulfate levels (10 to 20 $\mu g/m^3$) were about 1.5 times higher in the preceding decade than in 1971. Thus, most families participating in the study had, by and large, probably been exposed to much higher pollution levels during the preceding decade than during the study period. This improvement in air quality resulted from stringent control efforts carried out by private enterprise and governmental agencies.

Median, ninetieth percentile, and maximum levels, indicative of short-term pollutant exposure during the 32-week study period, are given in Table 5.3.2. Similar values for the summer following the survey are included for comparison. The maximum allowable short-term (24-hour) air quality limits for sulfur dioxide and suspended particulates were not exceeded during the study. Peak exposures to sulfur dioxide (143 to 349 $\mu g/m^3$) and suspended nitrates (5 to 20 $\mu g/m^3$) were higher in the Intermediate exposure areas than in the Low area. Surprisingly, peak suspended sulfate exposures in the Low exposure area (32 to 51 $\mu g/m^3$) were similar to those in the Intermediate communities (28 to 42 $\mu g/m^3$). Intercommunity patterns for median 24-hour exposure levels paralleled annual average exposures, and ninetieth percentile values exhibited a community pattern similar to that of peak exposures. Seasonal factors were operative, with overall higher pollutant exposures occurring during fall and winter. Total suspended particulates showed less of a seasonal trend than the other measured pollutants.

Table 5.3.1. ESTIMATED ANNUAL AVERAGE LEVELS OF SELECTED AIR POLLUTANTS IN NEW YORK METROPOLITAN STUDY COMMUNITIES

| Pollutant and community | Years prior to study[a] |  | During 1971 |
|---|---|---|---|
|  | 6 to 10 (1961-65) | 1 to 5 (1966-70) |  |
| Sulfur dioxide |  |  |  |
| Low | <30 | <30 | 23 |
| Intermediate I | 431 | 256 | 51 to 63[b] |
| Intermediate II | 396 | 321 | 51 to 58[b] |
| Total suspended particulate |  |  |  |
| Low | <40 | <40 | 34 |
| Intermediate I | 201 | 97 | 63 to 84[b] |
| Intermediate II | 182 | 123 | 86 to 104[b] |
| Suspended sulfate |  |  |  |
| Low | ∿10 | ∿10 | 10.2 |
| Intermediate I | 20 | 10 | 13.2 |
| Intermediate II | 18 | 15 | 14.3 |
| Suspended nitrate |  |  |  |
| Low | NA[c] | NA | 1.9 |
| Intermediate I | NA | NA | 3.5 |
| Intermediate II | NA | NA | 4.1 |

[a] Study began in September 1970 and ended in May 1971.

[b] The upper end of the range represents analysis of samples made by the New York City Department of Air Resources. The low figure represents analysis of samples collected during the same period and at the same site then mailed to the Research Triangle Park laboratory in North Carolina.

[c] NA—not available.

## Characteristics of the Study Population

Intercommunity differences in socioeconomic status, indoor air pollution, and mobility were assessed (Table 5.3.3). The Low exposure community ranked between the two Intermediate communities when either of two indices of socioeconomic status was considered. Educational attainment of fathers was lower and relative crowding was more frequent in the Intermediate II community and least frequent in the Intermediate I community. Diminished social status was accompanied by a moderate increase in the proportion of parents who smoked cigarettes and a decrease in residential mobility. Almost all families in the two Intermediate communities utilized gas for cooking as compared to only three-fifths of families in the Low exposure community. Since the two Intermediate exposure areas differed only slightly in their pollution exposures, the respiratory disease experience of families from these two communities could reasonably be pooled. The pooled Intermediate grouping more closely matched the Low exposure area in socioeconomic status, smoking habits, and mobility. Smoking intensity, among those who smoked differed little between the communities.

Over 3000 subjects participated in the study; these subjects are distributed by family segment and study period in Table 5.3.4. The number of

### Table 5.3.2. DAILY AIR POLLUTANT EXPOSURE PATTERNS IN THREE NEW YORK COMMUNITIES DISTRIBUTED BY SEASON

| Pollutant and community | Daily pollutant levels, µg/m$^3$ | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Median | | | | 90th percentile | | | | Maximum | | | |
| | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 |
| Sulfur dioxide | | | | | | | | | | | | |
| Low | 13 | 18 | 15 | 8 | 45 | 76 | 45 | 21 | 131 | 125 | 114 | 52 |
| Intermediate I | 56 | 34 | 48 | 29 | 120 | 119 | 108 | 71 | 231 | 287 | 143 | 107 |
| Intermediate II | 64 | 35 | 19 | 21 | 178 | 162 | 104 | 58 | 268 | 349 | 219 | 123 |
| Total suspended particulates | | | | | | | | | | | | |
| Low | 32 | 29 | 29 | 34 | 59 | 56 | 53 | 58 | 97 | 134 | 91 | 104 |
| Intermediate I | 56 | 55 | 57 | 60 | 93 | 90 | 102 | 101 | 170 | 156 | 193 | 152 |
| Intermediate II | 57 | 75 | 77 | 78 | 118 | 121 | 128 | 118 | 216 | 188 | 217 | 211 |
| Suspended sulfates | | | | | | | | | | | | |
| Low | 11 | 9 | 6 | 9 | 23 | 18 | 16 | 22 | 48 | 51 | 32 | 42 |
| Intermediate I | 14 | 12 | 9 | 12 | 22 | 20 | 17 | 27 | 28 | 39 | 36 | 37 |
| Intermediate II | 14 | 14 | 10 | 14 | 22 | 23 | 22 | 32 | 36 | 35 | 42 | 53 |
| Suspended nitrates | | | | | | | | | | | | |
| Low | 1 | 1 | 1 | 2 | 3 | 2 | 2 | 4 | 3 | 6 | 4 | 6 |
| Intermediate I | 2 | 1 | 2 | 4 | 3 | 3 | 5 | 8 | 6 | 6 | 11 | 15 |
| Intermediate II | 1 | 2 | 3 | 4 | 3 | 4 | 9 | 9 | 5 | 6 | 20 | 14 |

### Table 5.3.3. PANEL CHARACTERISTICS

| Characteristic | Community | | | |
|---|---|---|---|---|
| | | Intermediate | | |
| | Low | I | II | Pooled |
| Fathers with high school diploma, % | 73 | 76 | 60 | 69 |
| Families with ≤1.0 person per room, % | 85 | 92 | 72 | 83 |
| Families using domestic gas, % | 60 | 95 | 98 | 96 |
| Current cigarette smokers, % | | | | |
| Mothers | 37 | 37 | 47 | 41 |
| Fathers | 45 | 47 | 53 | 50 |
| Families moved during previous 5 years, % | 36 | 50 | 38 | 45 |

families varied in each area, with the Intermediate I community having the largest number (241) and the Intermediate II community having the smallest number (187). The number of families considered in the analysis is somewhat lower due to attrition during the study and the restriction that excluded individuals unless they reported for every period within any study segment. Attrition was somewhat higher in the Low exposure community than in the two Intermediate communities.

## Acute Respiratory Disease Evaluation

### *Seasonal Variation*

The study, which began in late September 1970 and terminated in early May 1971, encompassed an entire respiratory illness season. The overall acute respiratory illness attack rates for each of the study communities were plotted to detect any epidemic wavelets, but none occurred

### Table 5.3.4. STUDY PARTICIPANTS INCLUDED IN ANALYSES DISTRIBUTED BY SEASON AND COMMUNITY

| Family segment and community | Period of study | | | | Participated during all periods |
|---|---|---|---|---|---|
| | Weeks 0-8 | Weeks 9-16 | Weeks 17-24 | Weeks 25-32 | |
| Fathers | | | | | |
| Low | 191 | 185 | 184 | 163 | 132 |
| Intermediate I | 231 | 232 | 227 | 218 | 179 |
| Intermediate II | 140 | 181 | 183 | 175 | 157 |
| Pooled Intermediate | 371 | 413 | 410 | 393 | 336 |
| Mothers | | | | | |
| Low | 211 | 210 | 206 | 186 | 146 |
| Intermediate I | 238 | 241 | 235 | 224 | 183 |
| Intermediate II | 147 | 187 | 190 | 184 | 161 |
| Pooled Intermediate | 385 | 428 | 425 | 408 | 344 |
| School children | | | | | |
| Low | 564 | 561 | 551 | 492 | 388 |
| Intermediate I | 536 | 525 | 516 | 501 | 406 |
| Intermediate II | 331 | 401 | 423 | 403 | 349 |
| Pooled Intermediate | 867 | 926 | 939 | 904 | 755 |
| Preschool children | | | | | |
| Low | 170 | 135 | 133 | 122 | 84 |
| Intermediate I | 148 | 149 | 144 | 145 | 119 |
| Intermediate II | 95 | 121 | 125 | 122 | 97 |
| Pooled Intermediate | 243 | 270 | 269 | 267 | 216 |
| Total | | | | | |
| Low | 1136 | 1091 | 1074 | 963 | 750 |
| Intermediate I | 1153 | 1147 | 1122 | 1088 | 887 |
| Intermediate II | 713 | 890 | 921 | 884 | 764 |
| Pooled Intermediate | 1866 | 2037 | 2043 | 1972 | 1651 |

(Figure 5.3.1). Rates were high throughout fall and winter before declining in the spring. The attack rates for the two Intermediate areas were generally above that of the Low pollution area. Higher illness rates in the Intermediate I area were reported, despite the fact that current pollution levels were generally somewhat lower than those reported in the Intermediate II area. The discrepancy possibly was related to the additional pollution associated with the nearby airport, but this could not be substantiated with the data collected.

To assess the effect of pollution on interseasonal variations in respiratory disease, the time span of study was divided into roughly equal seasonal segments:

1. Season I, September 27 to November 21, 1970.
2. Season II, November 22, 1970, to January 16, 1971.
3. Season III, January 17 to March 13, 1971.
4. Season IV, March 14 to May 8, 1971.

Significant intercommunity differences in illness frequency were found in every season (Table 5.3.5). Illnesses involving the lower respiratory tract were significantly increased when residentially stable families (i.e., families who had not moved within the past 5 years) living in the two Intermediate communities were compared to families living in the Low

**Table 5.3.5. STATISTICAL ANALYSIS OF EFFECTS OF AIR POLLUTION ON FREQUENCY OF ILLNESS BY SEASON**[a]

| | Probability (p)[b] | | |
|---|---|---|---|
| Season | Upper tract illness | Lower tract illness | All respiratory illness |
| I | 0.01 | 0.005 | 0.001 |
| II | NS | 0.001 | 0.001 |
| III | NS | 0.01 | 0.10 |
| IV | NS | 0.025 | NS |

[a] Analysis included all members of residentially stable families, i.e., those who had not moved within the past 5 years.

[b] NS—not significant, $p > 0.10$.

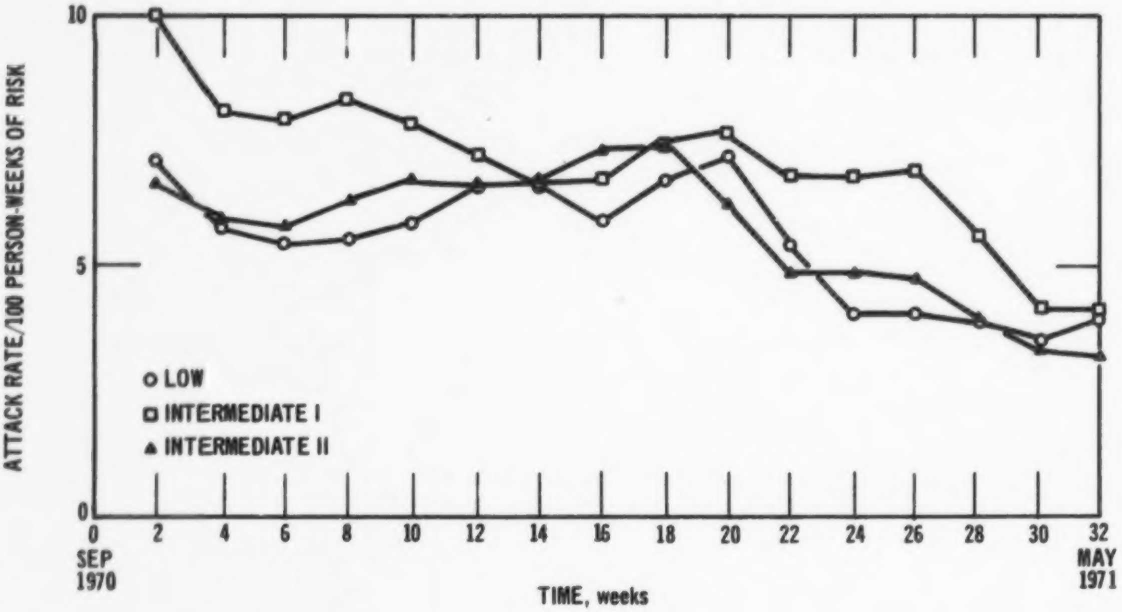

Figure 5.3.1. Attack rates for acute respiratory disease among families living in communities with differing air pollution exposures.

exposure community. Acute upper respiratory illnesses were significantly increased in the Intermediate communities only during the initial seasonal segment, and the greatest increases in overall acute respiratory illness occurred during the first two seasonal segments.

### *Illness Frequency*

For each community, overall acute respiratory disease attack rates were computed separately for fathers, mothers, elementary school children, and preschool children. To facilitate intercommunity comparisons, relative risk ratios were calculated, utilizing the experience of the Low exposure community as a base. The relative risk for any respiratory illness category involving family members from the Low exposure community was set at 1.00, and the relative risks for other communities were calculated by using the attack rate for the Low exposure community as the denominator. Sets of relative risks were compiled for each family segment and for each class of respiratory illness (Table 5.3.6). Because the classification of data obtained always categorized disease as lower tract illness if a single lower tract symptom was reported, the overall rates for lower tract illnesses were somewhat higher than those for upper tract illnesses—a condition recognized as being contrary to that observed in other studies. This factor should not affect the significance of the study results, however, since data were collected in the same manner in each community. Relative risks were also computed for the pooled Intermediate exposure group, which closely mimicked the socioeconomic characteristics of the Low exposure area. The tabulated base rates must be interpreted with caution since they consider only the 32-week season of greatest respiratory illness. The base rate expressed in illnesses per 100 person-weeks of exposure, in fact, roughly approximates the acute respiratory illness experience of the 32-week-long seasons, not the experience of a single year.

Table 5.3.6. RELATIVE RISK OF ACUTE RESPIRATORY ILLNESS

| Family segment and community | Relative risk of acute respiratory illness[a] Involving upper tract | Involving lower tract | All acute respiratory illness |
|---|---|---|---|
| Fathers | | | |
| Low | 1.00 (1.64) | 1.00 (1.80) | 1.00 (3.44) |
| Intermediate I | 0.94 | 1.38 | 1.17 |
| Intermediate II | 0.65 | 1.08 | 0.88 |
| Pooled Intermediate | 0.82 | 1.25 | 1.05 |
| Mothers | | | |
| Low | 1.00 (2.17) | 1.00 (2.28) | 1.00 (4.45) |
| Intermediate I | 0.97 | 1.30 | 1.14 |
| Intermediate II | 0.94 | 1.16 | 1.05 |
| Pooled Intermediate | 0.96 | 1.23 | 1.10 |
| School children | | | |
| Low | 1.00 (2.70) | 1.00 (3.51) | 1.00 (6.22) |
| Intermediate I | 1.04 | 1.31 | 1.20 |
| Intermediate II | 0.94 | 1.10 | 1.03 |
| Pooled Intermediate | 1.00 | 1.22 | 1.13 |
| Preschool children | | | |
| Low | 1.00 (3.30) | 1.00 (4.59) | 1.00 (7.88) |
| Intermediate I | 1.04 | 1.63 | 1.40 |
| Intermediate II | 0.87 | 1.15 | 1.03 |
| Pooled Intermediate | 0.97 | 1.42 | 1.20 |

[a] Base rates in parentheses refer to illnesses per 100 person-weeks at risk.

Intercommunity differences in relative risks were apparent, with the Intermediate communities consistently reporting significant increases in lower respiratory tract illness and total acute respiratory illness when compared with the Low exposure community (Table 5.3.7). The most striking finding was a marked (22 to 43 percent) excess risk for acute lower respiratory illness among families in the pooled Intermediate communities. In general, the Intermediate II community reported lower rates than did the Intermediate I community. Although upper respiratory illness was somewhat reduced among families living in the Intermediate areas, the general trend for total acute respiratory disease seemed to be more illness in these more polluted areas. The reduction in upper tract illness was not statistically significant.

**Table 5.3.7. STATISTICAL ANALYSIS OF EFFECTS OF AIR POLLUTION ON FREQUENCY OF ILLNESS BY FAMILY SEGMENT[a]**

| Family segment | Probability (p)[b] | | |
|---|---|---|---|
| | Upper tract illness | Lower tract illness | All respiratory illness |
| All members | NS | 0.005 | 0.025 |
| Adults | NS | 0.001 | 0.005 |
| Children | NS | 0.07 | NS |

[a] Statistical analysis was restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

[b] NS—not significant, $p > 0.10$.

### *Illness Severity*

Each illness was assigned a severity score based upon the previously defined five-level severity scale. To facilitate intercommunity comparisons, sets of relative severity scores were calculated for each acute respiratory illness class and for each family segment (Table 5.3.8). The

**Table 5.3.8. RELATIVE SEVERITY SCORES FOR ACUTE RESPIRATORY ILLNESS**

| Family segment and community | Relative severity score[a] | | |
|---|---|---|---|
| | Upper respiratory illnesses | Lower respiratory illnesses | All acute respiratory illnesses |
| Fathers | | | |
| Low | 1.00 (1.38) | 1.00 (1.76) | 1.00 (1.58) |
| Intermediate I | 1.19 | 1.07 | 1.13 |
| Intermediate II | 0.98 | 1.13 | 1.11 |
| Pooled Intermediate | 1.10 | 1.10 | 1.12 |
| Mothers | | | |
| Low | 1.00 (1.55) | 1.00 (1.92) | 1.00 (1.74) |
| Intermediate I | 1.19 | 1.14 | 1.21 |
| Intermediate II | 1.04 | 1.13 | 1.09 |
| Pooled Intermediate | 1.14 | 1.14 | 1.16 |
| School children | | | |
| Low | 1.00 (2.11) | 1.00 (2.28) | 1.00 (2.16) |
| Intermediate I | 1.17 | 1.22 | 1.21 |
| Intermediate II | 1.09 | 1.13 | 1.12 |
| Pooled Intermediate | 1.14 | 1.18 | 1.17 |
| Preschool children | | | |
| Low | 1.00 (2.21) | 1.00 (2.39) | 1.00 (2.31) |
| Intermediate I | 1.19 | 1.12 | 1.29 |
| Intermediate II | 1.09 | 1.16 | 1.21 |
| Pooled Intermediate | 1.15 | 1.14 | 1.26 |

[a] Base severity score in parentheses.

procedure was identical to that utilized in the relative risk model, except that the base was a severity score not an attack rate. Severity scores for all categories of respiratory illness were consistently elevated in the Intermediate communities, with the single exception of upper respiratory tract illnesses among fathers in the Intermediate II community. The greatest increases in relative severity scores (7 to 22 percent) occurred with acute lower respiratory illnesses. Statistical analysis for all family members, restricted to residentially stable families, indicated that increases in upper, lower, and all respiratory illness were statistically significant ($p < 0.01$, 0.001, and 0.001, respectively).

The average number of restricted activity days was then calculated for upper and lower tract illnesses falling within a given severity score (Table 5.3.9). There were some anomalous findings: for parents, school children, and preschool children increasing severity scores for upper tract illnesses from Level II to Level III reduced rather than increased the number of restricted activity days, and for preschool children a similar decrease was found for lower tract illness. One explanation might be that febrile upper tract illnesses are more often bacterial and thus respond more readily to antimicrobial therapy than do the more lingering infections produced by other agents. Despite these anomalies, the severity score proved a useful concept.

By applying mean severity scores to community-specific attack rates for each family segment, one could calculate the excess percentage of restricted activity days that might be attributable to living in one of the two Intermediate communities (Table 5.3.10). Direct estimates of yearly morbidity excesses cannot be made from Table 5.3.10 because results are expressed

**Table 5.3.9. AVERAGE NUMBER OF RESTRICTED ACTIVITY DAYS ACCOMPANYING EACH SEVERITY SCORE FOR ACUTE RESPIRATORY ILLNESS[a]**

| Family segment and severity score[b] | Mean number of restricted activity days at each level of severity | | |
|---|---|---|---|
| | Upper tract illness | Lower tract illness | All illness |
| Parents | | | |
| I | 0 | 0 | 0 |
| II | 3.17 | 3.59 | 3.41 |
| III | 2.44 | 3.92 | 3.42 |
| IV | 4.36 | 5.34 | 5.12 |
| School children | | | |
| I | 0 | 0 | 0 |
| II | 2.72 | 3.22 | 3.01 |
| III | 2.44 | 3.91 | 3.34 |
| IV | 4.82 | 5.73 | 5.39 |
| V | 4.50 | 5.96 | 5.45 |
| Preschool children | | | |
| I | 0 | 0 | 0 |
| II | 3.63 | 3.77 | 3.73 |
| III | 2.91 | 3.15 | 3.10 |
| IV | 4.29 | 5.63 | 5.31 |
| V | 4.89 | 6.28 | 5.82 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

[b] Severity score: I - illness without restricted activity, II - restricted activity but not fever, III - restricted activity and fever, IV - all of the preceding plus a physician visit, and V - restricted activity, fever, and otitis media diagnosed by a physician (limited to children).

Table 5.3.10. EXCESS RESTRICTED ACTIVITY DAYS NECESSITATED BY MORE FREQUENT OR MORE SEVERE ACUTE RESPIRATORY ILLNESS

| Family segment | Excess restricted activity days per 100 person-weeks of risk | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract illness | | Lower tract illness | | All illness | |
| | Percent excess | Excess days | Percent excess | Excess days | Percent excess | Excess days |
| Fathers | 12 | 0.24 | 55 | 2.70 | 42 | 2.94 |
| Mothers | 34 | 1.30 | 58 | 4.35 | 50 | 5.65 |
| School children | -3 | -0.21 | 33 | 3.97 | 20 | 3.76 |
| Preschool children | -8 | -0.88 | 33 | 5.50 | 17 | 4.62 |
| Nuclear family | 2 | 0.45 | 40 | 16.52 | 26 | 16.97 |

in excess restricted activity days per 100 person-weeks of risk. These rates were themselves based on the 32-week-long season of greatest respiratory illness. A better estimate of the excess number of days of restricted activity due to respiratory disease per 100 person-weeks of risk for a single year might be obtained by dividing the tabulated number of excess restricted activity days by three. The tabulated percentage excesses, however, should be relatively consistent over the shorter or longer period. The yearly excess morbidity is impressive and would involve 1 to 2 extra days of restricted activity every year for each member of the family. A nuclear family with two children residing in the Intermediate communities might experience 5.67 extra days of restricted activity, or 26 percent more restricted activity than an identical family living in the Low exposure community. The greatest percentage increase in restricted activity was occasioned by acute lower respiratory illness in adults; the greatest increase in actual days of illness occurred among preschool children.

Families living in the Intermediate communities also indicated that their illnesses were more severe by reporting that a higher percentage of their acute respiratory illnesses required restricted activity or a physician visit (Table 5.3.11), as might be reasoned from the relative severity scores. The prevalence of otitis media, which was another index of severity, did not differ between communities. By applying community-specific physician utilization rates to community-specific attack rates, estimates of excess physician visits attributable to residence in the Intermediate communities can be derived (Table 5.3.11). Among the volunteer families, the combination of more illness and more severe illness almost doubled physician visits resulting from acute respiratory illness. Clearly, the estimated increases in physician utilization and restricted activity constitute a potentially costly public health problem.

### *Vulnerability to Repeated Acute Respiratory Illness*

During the study, family members who reported more than two upper or two lower respiratory illnesses were judged to have repeated excessive illness. When the illness experience of residentially stable families in the study communities was reviewed, excessive repeated total acute respiratory illness was somewhat more common in children from more polluted communities (Table 5.3.12). However, no trend towards intercommunity differences was noted for acute lower respiratory disease, which might more likely be accompanied by an increased long-term risk for chronic lower respiratory disease.

### *Validity of the Analyses*

Additional analyses were undertaken to determine if the previously discussed relationships

Table 5.3.11. SEVERITY OF ACUTE RESPIRATORY ILLNESS[a]

| Family segment and community | Acute respiratory illness | | | | | | |
|---|---|---|---|---|---|---|---|
| | Percent requiring restricted activity | | | Percent requiring physician visit | | | |
| | Upper tract | Lower tract | Total | Upper tract | Lower tract | Total | Percent accompanied by otitis media |
| Fathers | | | | | | | |
| Low | 30 | 32 | 31 | 4 | 5 | 5 | -- |
| Pooled Intermediate | 29 | 49 | 41 | 4 | 12 | 9 | -- |
| Mothers | | | | | | | |
| Low | 28 | 46 | 29 | 3 | 13 | 8 | -- |
| Pooled Intermediate | 45 | 61 | 54 | 5 | 16 | 11 | -- |
| School children | | | | | | | |
| Low | 51 | 60 | 56 | 14 | 14 | 14 | 3.7 |
| Pooled Intermediate | 68 | 76 | 73 | 22 | 30 | 26 | 3.5 |
| Preschool children | | | | | | | |
| Low | 39 | 59 | 53 | 15 | 21 | 19 | 7.2 |
| Pooled Intermediate | 63 | 74 | 70 | 23 | 30 | 28 | 5.6 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

Table 5.3.12. REPEATED ACUTE RESPIRATORY ILLNESSES[a]

| Family segment and community | Percent of members with repeated acute respiratory illness | | |
|---|---|---|---|
| | > two upper tract illnesses | > two lower tract illnesses | > four total illnesses |
| **Fathers** | | | |
| Low | 9 | 5 | 4 |
| Pooled Intermediate | 2 | 5 | 2 |
| **Mothers** | | | |
| Low | 4 | 2 | 1 |
| Pooled Intermediate | 5 | 7 | 4 |
| **School children** | | | |
| Low | 10 | 16 | 10 |
| Pooled Intermediate | 11 | 15 | 12 |
| **Preschool children** | | | |
| Low | 2 | 23 | 16 |
| Pooled Intermediate | 14 | 28 | 25 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

between air pollutants and acute respiratory illness could be resulting spuriously from the effects of differences caused by reporting errors, socioeconomic factors, cigarette smoking, or familial susceptibility. The first factor investigated was the quality of respondent information. Greater than 90 percent reproducibility was found for the fact of acute respiratory illness, and there was greater than 98 percent reproducibility for the fact of no new illness. No intercommunity differences in the quality of respondent information were found.

Residential mobility determines pollution exposures and may itself influence the incidence of acute illness since changes in life situations seem to increase morbidity from diverse causes.[13] In the present study, residentially stable families were defined as those who had not changed address during the preceding 5 years. Some of the mobility was more apparent than real as some movement likely took place within a community rather than across communities. This was especially true in the Intermediate I community. Mobility-specific acute respiratory illness attack rates and appropriate sets of relative risks were calculated by the procedures previously described (Table 5.3.13). Mobility-specific total acute respiratory illness rates formed a consistent pattern: adults who recently moved to the Low exposure community reported acute lower respiratory illness rates much like those reported in the Intermediate communities. Since most of the families moving into the suburban fringe area were migrating from urban core areas, this residual susceptibility might be attributed to previous long-term air pollution exposures. In contrast, school children moving into the Low community reported lower respiratory illness rates about half way between those of the Intermediate and Low communities. Furthermore, preschool children who were recent migrants to the Low pollution area reported no excess lower respiratory disease, but did report higher rates of upper respiratory illness than any other group, migrant or stable. These findings could be interpreted as indicating that air pollution control should most quickly benefit children. Mobility-specific rates reinforced the observation that large increases in acute respiratory illness, principally lower tract illness, could be attributed to ambient air pollution exposure. The overall effect of including more mobile families in the initial

**Table 5.3.13. ACUTE RESPIRATORY ILLNESS AMONG STABLE AND MOBILE FAMILIES[a]**

| Family segment and community | Relative risk of acute respiratory illness[b] | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract illness | | Lower tract illness | | All respiratory illness | |
| | Mobile | Stable | Mobile | Stable | Mobile | Stable |
| Fathers | | | | | | |
| Low | 1.00(1.77) | 0.85 | 1.00(1.66) | 1.27 | 1.00(3.43) | 0.95 |
| Pooled Intermediate | 0.95 | 0.65 | 1.39 | 1.35 | 1.16 | 0.89 |
| Mothers | | | | | | |
| Low | 1.00(2.51) | 1.04 | 1.00(1.80) | 1.67 | 1.00(4.31) | 1.30 |
| Pooled Intermediate | 0.91 | 0.83 | 1.56 | 1.12 | 1.18 | 1.00 |
| School children | | | | | | |
| Low | 1.00(2.80) | 0.93 | 1.00(3.25) | 1.08 | 1.00(6.06) | 1.00 |
| Pooled Intermediate | 1.09 | 0.94 | 1.23 | 1.21 | 1.16 | 1.18 |
| Preschool children | | | | | | |
| Low | 1.00(2.71) | 1.48 | 1.00(5.47) | 0.73 | 1.00(8.18) | 0.98 |
| Pooled Intermediate | 1.26 | 1.19 | 1.25 | 1.21 | 1.25 | 1.15 |

[a] Stable families had lived at the same address during the 5 years preceding the study; mobile families had moved during the 5-year period.

[b] Base rate per 100 person-weeks of risk in parentheses.

**Table 5.3.14. EFFECT OF SOCIOECONOMIC STATUS UPON ACUTE RESPIRATORY ILLNESS**[a]

| Family segment and community | Relative risk of acute respiratory illness[b] | | | | | |
|---|---|---|---|---|---|---|
| | Upper tract illness | | Lower tract illness | | All illness | |
| | Higher status[c] | Lower status | Higher status[c] | Lower status | Higher status[c] | Lower status |
| Fathers | | | | | | |
| Low | 1.00(1.96) | 0.42 | 1.00(1.69) | 0.66 | 1.00(3.65) | 0.53 |
| Pooled Intermediate | 0.68 | 0.91 | 1.23 | 1.59 | 0.94 | 1.22 |
| Mothers | | | | | | |
| Low | 1.00(2.29) | 0.56 | 1.00(1.52) | 1.86 | 1.00(3.81) | 1.08 |
| Pooled Intermediate | 1.72 | 0.96 | 1.62 | 2.19 | 1.24 | 1.38 |
| School children | | | | | | |
| Low | 1.00(3.20) | 0.66 | 1.00(4.03) | 0.96 | 1.00(7.23) | 0.82 |
| Pooled Intermediate | 1.11 | 0.83 | 1.15 | 0.90 | 1.14 | 0.87 |
| Preschool children | | | | | | |
| Low | 1.00(3.21) | 0.41 | 1.00(6.90) | 0.64 | 1.00(10.12) | 0.57 |
| Pooled Intermediate | 1.04 | 1.72 | 1.14 | 0.48 | 1.10 | 0.87 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved in the past 5 years.

[b] Base rate per 100 person-weeks of risk in parentheses.

[c] Higher signifies homes with $\leq 1.00$ person per room.

analysis was to partially obscure the excess in illness that is most logically linked to air pollution exposures.

Differences in socioeconomic status were found to have a statistically significant ($p < 0.005$) effect on frequency of upper respiratory disease and total respiratory disease. Pooling the Intermediate exposure communities, however, should have minimized, if not eliminated, any bias due to social class difference. Socioeconomic-specific rates based on crowding in the home were computed for family segments in the Low exposure and pooled Intermediate exposure communities. Sets of relative risks were then calculated (Table 5.3.14). Families in the lower social category reported significantly less upper respiratory illness than did families in the higher social category. The differences in illness that were thought attributable to pollution exposure were not substantially diminished by comparison of socioeconomic-specific rates.

Cigarette smoking constitutes an intense self-pollution hazard, as well as contributing to indoor air pollution for other members of the family who do not themselves smoke. Pooling the two Intermediate communities left a small residual excess of smokers in the more polluted communities. Statistical analysis (Table 5.3.15) indicated that both personal smoking and smoking in the home significantly affected frequency of upper and lower respiratory illness, though not total respiratory illness. The indicated decrease in upper tract illness may be associated with the study design, which arbitrarily assigned the lower tract classification when both upper and lower tract symptoms were present. To evaluate these effects, smoking-specific attack rates were calculated for acute lower respiratory illness, and relative risk models were computed for each family segment (Table 5.3.16). Among adults, current cigarette smokers seemed to experience more illness than lifetime nonsmokers. Exsmokers also appeared to have a greater relative risk when compared to lifetime nonsmokers. Assessing the effect of smoking in the home on children was more difficult. Attempts to construct dose-response gradients for smoking in the home were partially frustrated by small sample sizes. Families could easily be divided into those with no smoking in the home, those with smoking in the home, and an indeterminant group. Families were assigned to the indeterminant group if the mother was an exsmoker, if the father was a current smoker, or if someone else in the home smoked. Exsmoking mothers were placed in the indeterminant category because it was not known when mothers ceased to smoke or if they resumed the habit during the study. When the appropriate relative risk models for children were constructed, a significant increase in the risk of acute lower respiratory attack rates was noted (Table 5.3.17). The adjustment somewhat diminished the relative risk attributable to pollution in children but increased the comparable risk among adults. Clearly, there were pollution effects quite independent of self-pollution by cigarette smoking.

**Table 5.3.15. STATISTICAL ANALYSIS OF EFFECT OF SMOKING ON FREQUENCY OF ILLNESS[a]**

| Determinant | Family segment | Probability (p)[b] | | |
|---|---|---|---|---|
| | | Upper tract illness | Lower tract illness | All respiratory illness |
| Personal cigarette smoking | Adults | 0.005[c] | 0.005 | NS |
| Cigarette smoking in home | Children | 0.005[c] | 0.01 | NS |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

[b] NS - not significant, $p > 0.10$.

[c] Associated with a decrease in illness frequency.

**Table 5.3.16. EFFECT OF CIGARETTE SMOKING ON ACUTE LOWER RESPIRATORY ILLNESS[a]**

| Current smoking status | Relative risk of acute lower respiratory illness by community[b] Low | Pooled Intermediate |
|---|---|---|
| Fathers | | |
| Lifetime nonsmoker | 1.00 (1.67) | 0.96 |
| Exsmoker | 1.19 | 1.51 |
| Smoker | 0.78 | 1.47 |
| Mothers | | |
| Lifetime nonsmoker | 1.00 (0.98) | 2.09 |
| Exsmoker | 2.32 | 2.66 |
| Smoker | 2.08 | 3.35 |
| School children | | |
| No smoking in home | 1.00 (3.49) | 0.80 |
| Indeterminant | 0.82 | 1.49 |
| Smoking in home | 1.39 | 1.30 |
| Preschool children | | |
| No smoking in home | 1.00 (6.04) | 0.82 |
| Indeterminant | 1.10 | 1.16 |
| Smoking in home | 0.98 | 1.27 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

[b] Base rate per 100 person-weeks of risk is in parentheses.

**Table 5.3.17. SMOKING-ADJUSTED ACUTE LOWER RESPIRATORY ILLNESS ATTACK RATES[a]**

| Family segment and community | Relative rate of acute lower respiratory illness[b] |
|---|---|
| Fathers | |
| Low | 1.00 (1.58) |
| Pooled Intermediate | 1.41 |
| Mothers | |
| Low | 1.00 (1.72) |
| Pooled Intermediate | 1.55 |
| School children | |
| Low | 1.00 (3.97) |
| Pooled Intermediate | 1.09 |
| Preschool children | |
| Low | 1.00 (6.12) |
| Pooled Intermediate | 1.10 |

[a] Analysis restricted to residentially stable families, i.e., those who had not moved within the past 5 years.

[b] Base rate per 100 person-weeks of risk in parentheses.

The value of chronic bronchitis in parents as a predictor of childhood respiratory illness was then evaluated (Table 5.3.18). When study areas were pooled, the few children of parents with chronic respiratory disease were found to have excessive acute lower respiratory illness. This excess was most consistent in families who did not smoke cigarettes and was most frequent in the Intermediate communities, suggesting that air pollution may be an additive hazard to a familial predisposition involving vulnerability of the lower respiratory tract.

### *Age-specific Attack Rates*

Age-specific acute respiratory attack rates among children from residentially stable families were examined to determine if reasonable estimates might be made of the length of exposure necessary to increase the vulnerability of a child to acute lower respiratory illness (Figure 5.3.2). Children from the Low exposure community were found to have somewhat higher attack rates during the first year of life than those from the Intermediate communities, suggesting the possibility either that longer exposures were necessary to induce a state of increased vulnerability or that older cohorts were carrying a residual impairment imprinted during earlier, much higher exposures to air pollution. Significant excess illness occured in the children from the Intermediate communities at 2 to 4 years of age, suggesting that 1 to 3 years might be the exposure duration necessary to increase susceptibility. One other finding seems to be important. Intercommunity differences were obliterated when children first attended school, suggesting that intense exposure to respiratory agents can overwhelm any differences caused by more subtle environmental factors like community air pollution. Smaller pollution-related increases in attack rates reappeared during the last 4 years of elementary school.

**Table 5.3.18. RELATIONSHIP BETWEEN CHRONIC BRONCHITIS IN PARENTS AND ACUTE RESPIRATORY ILLNESS IN THEIR CHILDREN**

| Age of child | Bronchitis in either parent | Relative risk of acute lower respiratory illness[a]: Homes without smokers | Homes with smokers | All homes |
|---|---|---|---|---|
| School | No | 1.00 (3.60) | 1.00 (4.55) | 1.00 (4.14) |
| | Yes | 2.43 | 0.97 | 1.19 |
| Preschool | No | 1.00 (5.55) | 1.00 (5.78) | 1.00 (5.67) |
| | Yes | 1.56 | 1.54 | 1.55 |

[a] Base rate per 100 person-weeks of risk in parentheses.

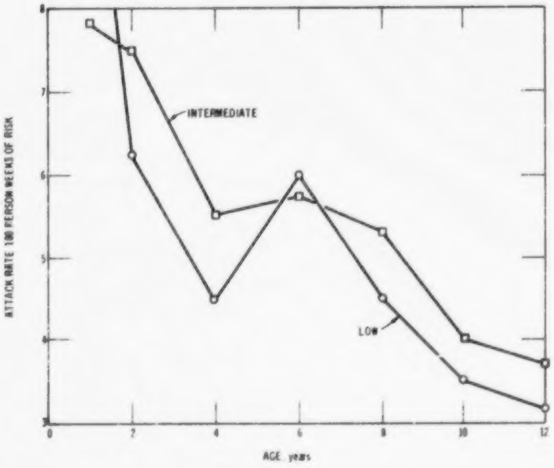

Figure 5.3.2. Age-specific attack rates for acute lower respiratory illness among children living in communities with differing air pollution exposure.

## DISCUSSION

The study demonstrated that acute lower respiratory tract illnesses are more frequent and more severe among families exposed to elevated levels of ambient air pollution. Evidence for increases in severity included additional days of restricted activity and additional physician visits. Residential mobility, socioeconomic status, cigarette smoking in the home, and family history of chronic bronchitis were all shown to be determinants of acute lower respiratory disease in children, but none of these factors accounted for the excesses attributed to ambient air pollution.

Two other intervening variables might alter the interpretation of the study: (1) indoor air pollution by the domestic use of gas for cooking or unvented space heating and (2) the presence of other not easily identified or unappreciated air pollutants. An attempt was made to disentangle the possible effects of cooking with gas from other determinants, but this was not possible because almost everyone in the Intermediate areas used gas and because sample sizes were very small when the experience of families in the Low exposure community was evaluated. The problem may be important since cooking with gas is alleged to generate substantial quantities of nitrogen dioxide, a pollutant that alters susceptibility to acute respiratory illness.[14,15] Unfortunately, no definitive answer can be given from the present study. Preliminary analysis did attribute a modest excess in the frequency of acute lower respiratory illness to indoor air pollution associated with the use of gas and indicated that this effect would diminish the excess in illness attributable to pollution by about 10 percent. On the other hand, adjustment for the use of gas would increase the frequency of acute upper respiratory disease attributable to pollution and leave unchanged the pollution-linked excesses found for total acute respiratory illness.

Other pollutants were monitored in the communities during or after the study now reported. Metal concentrations measured in dustfall and high-volume samples showed an increase in the Intermediate areas. Respirable particulate levels were found to parallel and to constitute roughly two-thirds of the total suspended particulate level. The Intermediate I exposure area, which manifested the highest illness rates, is located only a short distance from a large international airport and may be exposed to as yet undefined pollutants arising from aircraft emissions

Despite these uncertainties, we concluded that increases in acute lower respiratory disease morbidity can be attributed to exposures of 2 to 3 years involving annual average sulfur dioxide levels of 256 to 321 $\mu g/m^3$ accompanied by elevated annual average levels of total suspended particulate of 97 to 123 $\mu g/m^3$ and annual average suspended sulfate levels of 10 to 15 $\mu g/m^3$. There is a distinct possibility that increased susceptibility to acute lower respiratory illness is maintained or even induced by substantially lower ambient air pollution exposures involving annual average sulfur dioxide levels of 51 to 63 $\mu g/m^3$ accompanied by annual average total suspended particulate levels of 63 to 104 $\mu g/m^3$ and annual average suspended sulfate levels of 13 to 14 $\mu g/m^3$.

## SUMMARY

A prospective survey of over 3000 volunteers from families living in three communities with differing air pollution exposures attributed significant increases in the frequency and severity of acute lower respiratory illnesses to ambient air pollutants. Exposures of 2 to 3 years involving 256 to 321 $\mu g/m^3$ of sulfur dioxide accompanied by 97 to 123 $\mu g/m^3$ of total suspended particulates and 10 to 15 $\mu g/m^3$ of suspended sulfates were linked to excessive illnesses. There was suggestive evidence that control of air pollution substantially decreases susceptibility to acute lower respiratory illness. The average family of four might expect to experience 5 extra days of restricted activity and one extra physician visit each year as a result of the excess acute lower respiratory disease morbidity. Illness frequency was found to increase with increasing socioeconomic status, recent family migration, cigarette smoking in the home, and parental history of chronic bronchitis.

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# 5.4 AGGRAVATION OF ASTHMA BY AIR POLLUTANTS: 1970-1971 NEW YORK STUDIES

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## INTRODUCTION

The air pollutant levels that aggravate asthma and the complex relationships that determine the response of asthmatics are subjects of major public health significance because 3 to 5 percent of the U.S. population are asthmatics.[1,2] National Primary Ambient Air Quality Standards are based upon the need to minimize or prevent deleterious effects to the health of especially vulnerable population groups, including asthmatics.

Identification of the precipitating factors in most acute diseases is relatively easy. Asthmatic attacks, though capricious, are acute, and evidence of airborne substances being capable of precipitating an attack is well documented.[3-15] If short-term air pollutant exposures precipitate asthmatic attacks, then it should be possible to establish a dose-response relationship between asthma attack rates and pollutant levels. However, the effects of air pollutants must be separated from concurrent meteorological influences, from effluents of agricultural activity, and from pollutants peculiar to certain industries. Two additional demanding tasks are the need to separate the effects of one pollutant from those of another and the need to assure that human exposures are adequately monitored. Any single study may be unable to accomplish these goals, but a series of well-designed standardized studies separated in time and space can provide the needed health intelligence.

The study reported here had three specific goals: first, to ascertain, in a large urban area, which components of the ambient air pollution mixture might cause an excess in the asthma attack rates for panels composed of known asthmatics; second, to establish threshold levels for these pollutants; and third, to establish the relative importance of air pollutants and meteorologic factors. The study was conducted from October 1970 to May 1971 by the U.S. Environmental Protection Agency (EPA) with the cooperation of the New York City Department of Air Resources (NYC-DAR) and the Suffolk County (New York) Department of Health.

## METHODS

### The Study Population

Panelists were asthmatics who lived in three New York communities selected for EPA Community Health and Environmental Surveillance System (CHESS) studies because they had similar socioeconomic, racial, and ethnic makeup but differed in pollution exposure. NYC-DAR and the Suffolk County (New York) Department of Health guided the selection of the CHESS communities by furnishing historical air quality data and by facilitating the necessary air monitoring. After considering long-term air pollution exposure trends, Riverhead, Long Island, was chosen as a Low exposure community, the Howard Beach section of Queens as an Intermediate exposure community, and the Westchester section of the Bronx as a High exposure community. As a result of recent improvements in air quality, the Queens and Bronx were found to have similar current pollutant concentrations, often below National Primary Air Quality Standards. Hence, the latter two communities were redesignated Intermediate I and Intermediate II, respectively. The three communities chosen represent air pollution exposures involving sulfur dioxide, suspended sulfates, and total suspended particulates. Other pollutants are also present including nitrates, nitrogen dioxide, organics, metals, carbon monoxide, and gaseous hydrocarbons. The most recent census data, along with more current information on house and property values, supplied the socioeconomic profiles.

Rosters of possible asthmatic panelists were compiled from hospital clinic records and records of practicing physicians. Each prospective subject was interviewed by trained interviewers to obtain information regarding the nature, frequency, and severity of asthma. Smoking history, history of occupational exposure to respiratory irritants, and socioeconomic status were also determined.[16] Those subjects who had been diagnosed by a physician as being asthmatic and whose symptoms consisted of wheezing with an accompanying dyspnea were accepted if they had experienced three such episodes during the previous year. Selection of subjects with these criteria plus general agreement on the part of the physicians as to a definition of asthma enabled a uniform selection of panelists. Only panelists who lived within a 1.5-mile radius of monitoring stations were enrolled. The three panels initially consisted of 50 subjects in the Intermediate I community, 50 in the Intermediate II community, and 52 in the Low community.

### Diary Coverage

Each panelist received a diary weekly by mail,[16] recorded attacks as they occurred each day, and returned the completed diary by mail at the end of the week. When instructions for completing the diary were given by the interviewers, each panelist was asked to indicate on the diary that he had an attack whenever he became aware of shortness of breath combined with wheezing. Nonresponse and diaries requiring clarification were investigated by telephone. Diaries received more than 12 days after the last day covered by the diary and diaries that could not be corrected were not accepted for data processing. A log book was maintained to permit constant evaluation of each panelist's performance. Diary entries were transferred to the panelist's individual log page each week. The log book was reviewed weekly to identify over- and underreporting of asthma attacks, which might indicate misunderstanding of what was to be reported, to look for repeated corrections of diaries, which were usually due to a misunderstanding of the diary method, and to evaluate frequency of failure to return diaries. Appropriate contact was made with the panelists to improve performance, and continued nonresponders were dropped from the study.

## Monitoring Air Pollution Exposures

Air monitoring stations were located in each community on the roof of a two- or three-story building 30 to 45 feet above the ground. Measurement devices at each station included high-volume samplers for suspended particulates, dustfall buckets, and bubblers for nitrogen dioxide and sulfur dioxide. Continuous 24-hour monitoring was maintained throughout the study period. Sampler filters and bubblers were replaced daily, while dustfall buckets were replaced monthly. In the laboratory, sulfur dioxide and nitrogen dioxide were analyzed by standard EPA methods: West-Gaeke and Jacobs-Hochheiser, respectively. After dehydration, contents of the dustfall buckets were weighed. High-volume sampler filters were measured gravimetrically and analyzed for sulfate and nitrate fractions. The latter was determined by a reduction-diazo coupling method with automated analysis. For the sulfate fraction, a turbidimetric method was used, and turbidity was measured by spectrophotometer. A more detailed description of air monitoring methods is presented elsewhere.[17] Continuous measurements of temperature were obtained for the duration of the study from airports near the study communities.

Recent reevaluation by EPA indicates that the measurement method used for nitrogen dioxide is subject to interferences and variable collection efficiency. Therefore, the nitrogen dioxide data, while still included elsewhere,[17] are not included in this paper. The measurements made, however, indicated that nitrogen dioxide levels in the three areas were relatively low.

## Statistical Analysis

Daily asthma attack rates were computed by dividing the number of panelists who reported one or more asthma attacks on a given day by the number who returned their diaries for that day. This rate computation method was used because some panelists dropped from the study during the study period and others were added and because some diaries were not returned by panelists who traveled away from the area or who were occasionally forgetful.

The data for a single day of the study, then, consisted of the following items: a daily asthma attack rate, 24-hour averages of the measurements of selected pollutants, and a minimum and a maximum temperature. These data items for each community were available for each day of the study with the exception of some missing data for the pollutants. In all analyses, the asthma attack rate was considered the response variable and the others the independent variables.

The statistical analysis involved five sequential steps: first, plotting weekly averages of asthma attack rates, pollutant levels, and minimal temperature; second, calculating a simple correlation matrix for daily asthma attack rates, pollutant levels, and minimum temperature; third, performing multiple regression analyses to look for pollutant effects after removal of temperature effects; fourth, constructing temperature-specific relative risk and excess risk models for various pollutant levels; and fifth, computing temperature-specific dose-response estimates to locate threshold levels for pollutant effects.[18-20]

To calculate relative risk functions, the total experience of all four panels was combined into a series of temperature-specific relative risk models for appropriate concentrations of each of the pollutants of interest. Since the panels differed to some extent from each other in the intrinsic frequency of asthmatic attacks, a separate temperature-specific relative risk for each was computed based upon the experience of the panel during the days falling in the lowest concentration range for the particular pollutant of interest. Then, for each panel, relative risks for each subsequent pollutant level in the temperature-specific series were computed using the rate from the lowest pollutant level as the denominator. Relative risks were then weighted by the number of person-days of risk that each represented and combined across communities to achieve a single pooled rate for each temperature-specific pollutant level. The person-days of risk associated with any pooled rate were tallied so that an idea of rate stability might be conveyed. Because the analyses included days on which one or another pollutant might be missing in one or another community, temperature-specific groups of person-days will not balance.

Threshold functions were computed by the method of Hasselblad et al.[19] In setting air quality standards, the existence of a threshold concentration for a health effect is implicitly assumed. It is important, therefore, to ascertain if the dose-response relationship can be reasonably estimated by a function indicating no response until some nonzero threshold concentration is exceeded. Most statistical procedures assume a strictly monotonic functional relationship between two variables, and therefore, a significant association exists at all pollutant levels, no matter how low.

As a simple alternative, we hypothesized, for a threshold function, a segmented line having zero slope below exposure level x and positive slope at levels above x. The point x is estimated by the least squares method. This function has been designated a "hockey stick" function.[19] This least squares technique had been earlier applied to other more general problems.[20]

The estimated threshold, estimated slope, estimated intercepts, and the assumption of a linear response above the threshold permitted calculation of an excess risk at the current short-term air quality standard. Multiplying the difference between the standard and the threshold by the estimated slope of the response function provided the estimated asthma attack rate at these levels presently allowable once per year. Percentage of excess risk was calculated by dividing this estimated attack rate by the estimated attack rate intercept and then subtracting one.

## RESULTS

### Environmental Exposures

Fluctuations in short-term peak pollutant levels would be more likely to cause fluctuations in asthma attack rates than would exposures over longer averaging times. The median, 90th percentile, and maximum 24-hour pollutant levels were tallied for each study community and for each season (Table 5.4.1). As expected, the Intermediate exposure areas were generally found to have higher values than the Low exposure community at all three points on the cumulative frequency distributions for each pollutant. Communities differed most markedly in exposures involving sulfur dioxide, total suspended particulates, and suspended nitrates. Relatively minor intercommunity differences were found for suspended sulfates because of unexpectedly high levels in the Low exposure community that may have resulted from windborne transport of the pollutant into the suburban fringe community from more urbanized areas. Dustfall metals also differed, with higher levels of cadmium, lead, zinc, and other trace metals being found in the Intermediate exposure communities. Despite substantial differences in short-term air pollution exposure, no community reported sulfur dioxide concentrations as high as the 24-hour level (365 $\mu g/m^3$) that is allowable once per year under the relevant National Primary Air Quality Standard. Similarly, all communities met the short-term National Air Quality Standard for suspended particulates.

On the basis of annual average levels, the Low exposure community was well within all National Primary Ambient Air Quality Standards. The Intermediate exposure communities, while achieving substantial recent improvements in air quality, nevertheless exceeded the sulfur dioxide levels permitted by the National Secondary Air Quality Standard and the suspended particulate levels permitted by the National Primary Air Quality Standard. In summary, though air quality was substantially better on the suburban fringe than in the city itself, all three study communities were within or nearly within the National Ambient Air Quality Standards. Thus, any adverse health effect attributed to air pollutants

Table 5.4.1. DAILY AIR POLLUTANT EXPOSURE PATTERNS IN THREE NEW YORK COMMUNITIES DISTRIBUTED BY SEASON

| Pollutant and community | Daily pollutant levels, $\mu g/m^3$ | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Median | | | | 90th percentile | | | | Maximum | | | |
| | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 |
| Sulfur dioxide | | | | | | | | | | | | |
| Low | 13 | 18 | 15 | 8 | 45 | 76 | 45 | 21 | 131 | 125 | 114 | 52 |
| Intermediate I | 56 | 34 | 48 | 29 | 120 | 119 | 108 | 71 | 231 | 287 | 143 | 107 |
| Intermediate II | 64 | 35 | 19 | 21 | 178 | 162 | 104 | 58 | 268 | 349 | 219 | 123 |
| Total suspended particulates | | | | | | | | | | | | |
| Low | 32 | 29 | 29 | 34 | 59 | 56 | 53 | 58 | 97 | 134 | 91 | 104 |
| Intermediate I | 56 | 55 | 57 | 60 | 93 | 90 | 102 | 101 | 170 | 156 | 193 | 152 |
| Intermediate II | 57 | 75 | 77 | 78 | 118 | 121 | 128 | 118 | 216 | 188 | 217 | 211 |
| Suspended sulfates | | | | | | | | | | | | |
| Low | 11 | 9 | 6 | 9 | 23 | 18 | 16 | 22 | 48 | 51 | 32 | 42 |
| Intermediate I | 14 | 12 | 9 | 12 | 22 | 20 | 17 | 27 | 28 | 39 | 36 | 37 |
| Intermediate II | 14 | 14 | 10 | 14 | 22 | 23 | 22 | 32 | 36 | 35 | 42 | 53 |
| Suspended nitrates | | | | | | | | | | | | |
| Low | 1 | 1 | 1 | 2 | 3 | 2 | 2 | 4 | 3 | 6 | 4 | 6 |
| Intermediate I | 2 | 1 | 2 | 4 | 3 | 3 | 5 | 8 | 6 | 6 | 11 | 15 |
| Intermediate II | 1 | 2 | 3 | 4 | 3 | 4 | 9 | 9 | 5 | 6 | 20 | 14 |

would probably be occurring at levels below those currently thought to protect the public health.

## Characteristics of the Study Population

Although the analysis focused on the asthma attack experience of each panel over time rather than on intercommunity differences, every effort was made to select panels as alike as possible. The three panels were similar in number of participants, age, sex, smoking habit, and frequency of asthmatic attacks during the previous year (Table 5.4.2). The Intermediate II community was somewhat different in that fewer adult panelists were high school graduates. The significant difference in educational attainment of Intermediate II panelists might well affect reporting, but one would expect that the lower educational level of this panel might lead to underreporting for the self-administered questionnaires used and thus make associations with pollutants more difficult. About half of the panelists in each community gave a history of one or more nonasthmatic allergies including atopic dermatitis, allergic rhinitis, and urticaria. There were, however, no differences in the history of such allergic conditions or the duration of asthma itself. The overall response rate for the three panels was good; 75 percent of the more than 4700 diaries mailed were useable in the analysis. Weekly response rates varied somewhat for any particular week, but no notable intercommunity differences occurred.

## Temporal Patterns

Seasonal trends in asthma attack rates, pollutant levels, and minimum temperatures can help identify major determinants of illness that must be isolated or adjusted for in more detailed analyses. Seasonal trends were constructed separately for each community by plotting weekly averages for the variables. Asthma attack rates were plotted together with temperature and then in sequence with each of the air pollutants considered. All three communities were evaluated, but for illustrative purposes, only plots for the Low and Intermediate I communities are presented.

The temperature patterns were typical for the late fall, winter, and early spring months of

**Table 5.4.2. CHARACTERISTICS OF ASTHMA DIARY STUDY POPULATION**

| Population characteristics | Community | | | Total |
|---|---|---|---|---|
| | Low | Intermediate | | |
| | | I | II | |
| Number of subjects | 52 | 50 | 46 | 148 |
| Subjects > 16 years old, % | 50 | 50 | 46 | 48 |
| Female subjects, % | 48 | 38 | 43 | 43 |
| ≥High school completed by household head, % | 42 | 60 | 11 | 39 |
| Adults currently smoking, % | 19 | 16 | 38 | 12 |
| ≥10 asthma attacks during past year, % | 56 | 60 | 70 | 61 |

the study (Figure 5.4.1). The Low exposure community's asthma attack curve was highest in the early weeks of study, with a general downward trend thereafter. The attack rate curves of the Intermediate communities showed no overt seasonal trends. No clear association between temperature and asthma attack rates could be discerned in the plots for these variables. It should be noted, however, that asthma data were collected during a period characterized by a relatively narrow temperature band, when the majority of mean temperatures were near or below the freezing point.

No simple relationship linked sulfur dioxide levels with asthma attack rates (Figure 5.4.2). A possible temperature effect on sulfur dioxide levels can be seen: concentrations were highest in late fall and early winter when temperatures were at their lowest levels, low during the blustery weeks of late winter and early spring, and elevated again during April and May.

No seasonal trend was found for total suspended particulates (Figure 5.4.3). Weekly fluctuations in particulate levels were wider in the Intermediate community. The plots suggested a tenuous relationship between asthma attack rates and suspended particulates in the Intermediate community but not in the Low community.

Suspended sulfate levels proved quite variable in both communities, with the highest concentrations observed during the coldest weather (Figure 5.4.4). The plots suggested no overt relationship between sulfates and asthma attack rates in any community. When the seasonal plots were repeated for suspended nitrates (not illustrated), levels tended to be higher in the spring but not related to fluctuations in asthma attack rates.

## Correlation and Stepwise Multiple Regression Analyses

A complex web of constantly changing, interacting factors makes the separation of the underlying determinants of asthma attack rates difficult. One step in reducing the problem was to compute a simple correlation matrix to discern the relationships between temperature, asthma attack rate, and pollutant levels. When this was done, pollutants were generally found to be correlated with each other (Table 5.4.3); the single exception was the nonsignificant correlation between sulfur dioxide and suspended nitrates.

Pollutants varied in their relationship to temperature: suspended nitrates rose as temperature rose; total suspended particulates were little

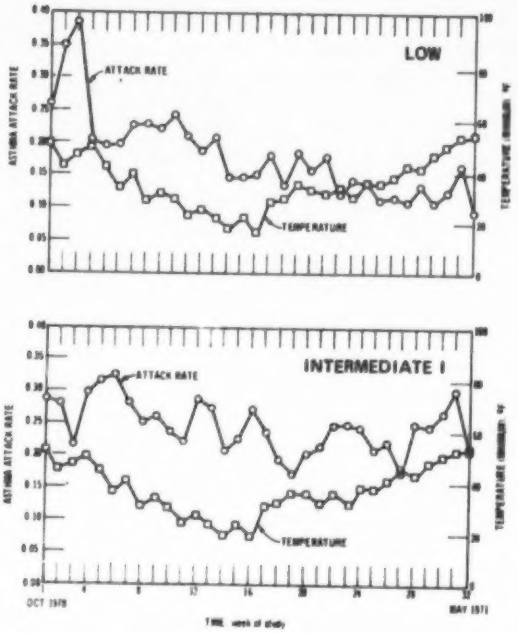

Figure 5.4.1. Weekly mean attack rates compared with weekly average minimum temperatures.

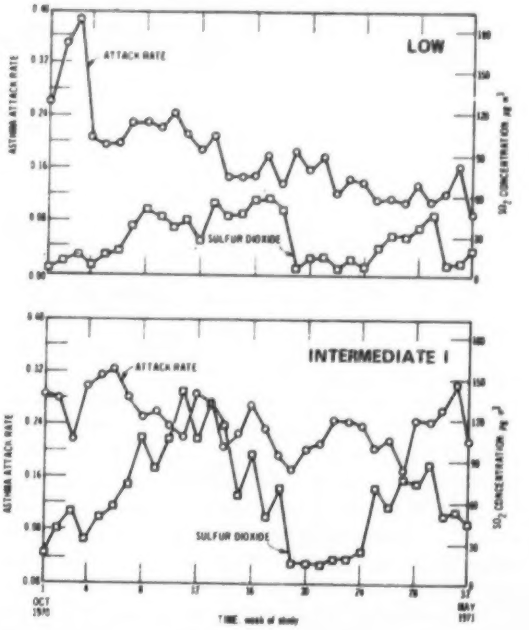

Figure 5.4.2. Weekly mean attack rates compared with weekly mean sulfur dioxide levels.

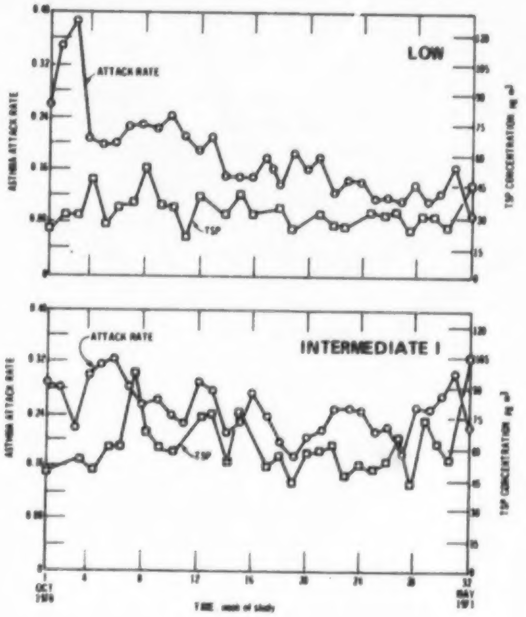

Figure 5.4.3. Weekly mean attack rates compared with weekly total suspended particulate levels.

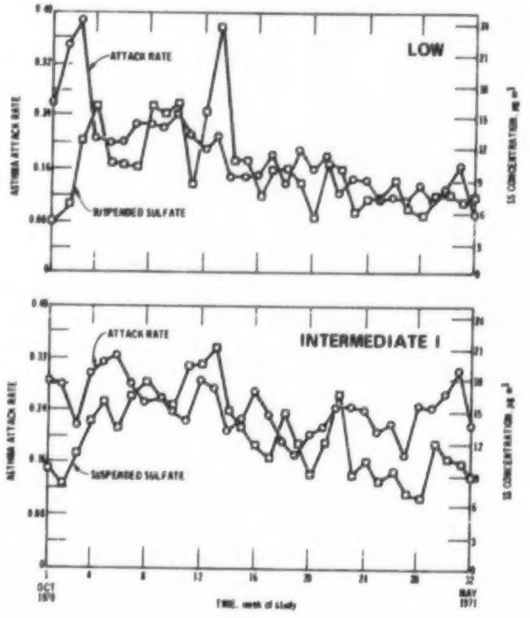

Figure 5.4.4. Weekly mean attack rates compared with weekly mean suspended sulfate levels.

**Table 5.4.3. SIMPLE CORRELATION COEFFICIENTS OF DAILY ATTACK RATES, POLLUTANTS, AND TEMPERATURE**

| Pollutant and community | Attack rate | TSP | SN | SS | $SO_2$ |
|---|---|---|---|---|---|
| Total suspended particulate | | | | | |
| Low | 0.058 | | | | |
| Intermediate I | 0.058 | | | | |
| Intermediate II | 0.064 | | | | |
| Suspended nitrates | | | | | |
| Low | 0.127 | 0.483[b] | | | |
| Intermediate I | 0.120 | 0.402[b] | | | |
| Intermediate II | 0.086 | 0.604[b] | | | |
| Suspended sulfates | | | | | |
| Low | 0.145[a] | 0.795[b] | 0.356[b] | | |
| Intermediate I | 0.106 | 0.506[b] | 0.112 | | |
| Intermediate II | -0.109 | 0.625[b] | 0.289[b] | | |
| Sulfur dioxide | | | | | |
| Low | -0.022 | 0.364[b] | 0.118 | 0.369[b] | |
| Intermediate I | 0.061 | 0.381[b] | 0.076 | 0.339[b] | |
| Intermediate II | -0.211[b] | 0.230[b] | -0.026 | 0.338[b] | |
| Minimum temperature | | | | | |
| Low | -0.002 | 0.006 | -0.078 | -0.153[a] | -0.226[b] |
| Intermediate I | 0.056 | 0.029 | 0.222[b] | -0.255[b] | -0.195[b] |
| Intermediate II | 0.177[a] | 0.075 | 0.258[b] | -0.241[b] | -0.365[b] |

[a] Significant at $p < 0.05$.

[b] Significant at $p < 0.001$.

altered by temperature fluctuations; and suspended sulfates and sulfur dioxide rose as temperatures fell, probably reflecting the use of fossil fuels for heating. It seemed possible to separate the effects of most single pollutants from the effects of other pollutants, with the exception of sulfur dioxide and suspended sulfates.

No consistent significant correlations were found between attack rates and any of the pollutants or between attack rates and temperature. A significant positive correlation of asthma attacks with ambient temperature was found for the Intermediate II community. It is difficult to interpret this relationship. It is also hard to explain the seemingly beneficial effect of elevated sulfur dioxide observed in the Intermediate II community.

Multiple regression analysis was used to clarify these relationships and to separate the effects of pollutants from those related to fluctuations in temperature (Table 5.4.4). First, temperature alone was considered; then the effect of pollutants after removal of any effects that could be related to temperature was tested. This analysis may be a conservative estimate of pollutant effects because some of the temperature effect may really be due to pollutants, such as sulfur dioxide and suspended sulfates, that are inversely correlated with temperature. In contrast to findings of other studies, no strong temperature effect was apparent. Suspended nitrates were linked weakly to excessive rates in the Low exposure area, perhaps reflecting their own correlation with sulfates. More importantly, multiple regression analyses unmistakably linked elevated suspended sulfates with excessive asthma and

**Table 5.4.4. SUMMARY OF MULTIPLE REGRESSION ANALYSIS CONSIDERING EFFECT OF MINIMUM TEMPERATURE FOLLOWED BY EFFECTS OF AIR POLLUTANTS**[a]

| Source of variation | Community | | |
|---|---|---|---|
| | | Intermediate | |
| | Low | I | II |
| Temperature alone | NS | NS | NS |
| Temperature adjusted | | | |
| $SO_2$ | NS | NS | $<0.02$ |
| TSP | NS | NS | NS |
| SS | $<0.05$ | $<0.10$ | NS |
| SN | $<0.10$ | NS | NS |

[a] NS—not significant, $p > 0.10$.

suggested that sulfur dioxide also contributed to increases in attack rates. Since sulfur dioxide, suspended particulates, and suspended sulfates are closely correlated with each other, and since two of the three had significant effects, it was decided to limit further analyses to these three pollutants.

## Temperature-specific Relative Risk Models

Linear functions predicted that temperature alone had little effect on asthma attack rates. Wide ranges in temperature were monitored during the study period, but there were fewer than 20 warm days ($T_{min} > 50$ °F). Perhaps, then, the present study was centered in a colder temperature range where there could be little or no further increases in asthma attributable to temperature alone. Temperature-specific relative risk models would avoid assumptions of a linear temperature effect and might allow separate estimates for the effects of pollutants on cold, freezing days ($T_{min} < 30$ °F) and on somewhat warmer days ($T_{min}$ = 30 to 50 °F).

In the relative risk models, a consistent temperature-related pattern appeared (Table 5.4.5). None of the pollutants seemed to have any measurable effect on asthma attack rates on very cold, freezing days. However, asthma attack rates for cleaner days at each temperature level were quite similar. When temperatures increased to the next level above freezing ($T_{min}$ = 30 to 50 °F) dose-related increments in asthma attack rates occurred for total suspended particulates and for suspended sulfates, but not for sulfur dioxide. The same pattern was apparent on the few warm days ($T_{min} > 50$ °F) included in the study. For temperatures above 30 °F, elevated asthma attack rates were noted for suspended particulate exposures of 76 to 260 $\mu g/m^3$ and for suspended sulfate exposures of 10 $\mu g/m^3$. Analysis of temperature-specific attack rates thus proved extremely helpful in identifying temperature ranges within which pollutant effects occurred, the general pollutant level accompanying the adverse health effect, and the magnitude of the effect. Increases as high as 13 percent in asthma attack rates can occur with particulate levels below the 24-hour level currently allowable once per year. A 10 percent increase in the risk of asthma occurred on days when suspended sulfate concentrations exceeded 10 $\mu g/m^3$. However, control of air pollution requires more precise estimates of response thresholds and dose-response relationships than are possible from the relative risk analyses employing temperature-specific attack rates.

## Threshold and Linear Dose-response Relationships

To determine the highest pollutant level that would fail to have an adverse effect on asthma attacks, the concurrent effects of ambient temperature and the limitations of the study were considered. The present study was restricted to a rather narrow range of sulfur dioxide and suspended particulate concentrations and focused upon cooler seasons, making threshold estimates for warmer days difficult. Sulfur dioxide levels exceeded 200 $\mu g/m^3$ and suspended particulate levels exceeded 150 $\mu g/m^3$ on less than 5 percent of the study days. Suspended sulfate levels ranged widely, with peak values over 50 $\mu g/m^3$. One might therefore expect that good threshold estimates would be limited to the lower temperature ranges and to suspended sulfates, with less of an opportunity to detect thresholds for suspended particulates or sulfur dioxide.

Threshold estimates were calculated for total suspended particulates and for suspended sulfates, but no linear or threshold effects were demonstrable for sulfur dioxide (Table 5.4.6). When days were cool, yet not frigid ($T_{min}$ = 30 to 50

### Table 5.4.5. AGGRAVATION OF ASTHMA QUANTIFIED BY TEMPERATURE-SPECIFIC RELATIVE RISK MODELS

| Pollutant and minimum temperature | Integrated 24-hour level, $\mu g/m^3$ | Relative risk[a] | Person-days of observation[b] |
|---|---|---|---|
| Sulfur dioxide | | | |
| <30 °F | ≤60 | 1.00 (21.0) | 2832 |
| | 61 to 80 | 0.99 | 515 |
| | 81 to 365 | 0.91 | 2228 |
| | >365 | – | – |
| 30 to 50 °F | ≤60 | 1.00 (22.2) | 10477 |
| | 61 to 80 | 1.06 | 1481 |
| | 81 to 365 | 0.98 | 2835 |
| | >365 | – | – |
| >50 °F | ≤60 | 1.00 (21.5) | 1902 |
| | 61 to 80 | 0.87 | 203 |
| | 81 to 365 | 0.97 | 287 |
| | >365 | – | – |
| Total suspended particulates | | | |
| <30 °F | ≤60 | 1.00 (21.1) | 4063 |
| | 61 to 75 | 0.97 | 898 |
| | 76 to 260 | 1.01 | 1351 |
| | >260 | – | – |
| 30 to 50 °F | ≤60 | 1.00 (21.3) | 10265 |
| | 61 to 75 | 1.02 | 1766 |
| | 76 to 260 | 1.13 | 2959 |
| | >260 | – | – |
| >50 °F | ≤60 | 1.00 (21.2) | 1545 |
| | 61 to 75 | 0.92 | 396 |
| | 76 to 260 | 1.05 | 451 |
| | >260 | – | – |
| Suspended sulfates | | | |
| <30 °F | ≤6.0 | 1.00 (19.1) | 433 |
| | 6.1 to 8.0 | 1.04 | 702 |
| | 8.1 to 10 | 1.02 | 1110 |
| | >10 | 0.97 | 4007 |
| 30 to 50 °F | ≤6 | 1.00 (19.0) | 2549 |
| | 6.1 to 8.0 | 1.04 | 2300 |
| | 8.1 to 10 | 1.09 | 2708 |
| | >10 | 1.08 | 7274 |
| >50 °F | ≤8.0 | 1.00 (22.1) | 915 |
| | 8.1 to 10 | 1.03 | 515 |
| | >10 | 1.10 | 1046 |

[a] Base rate for temperature-specific relative risk is in parentheses.

[b] Person-days for pollutants may not balance across temperature-days because one or more pollutants may be missing on one or more days.

**Table 5.4.6. TEMPERATURE-SPECIFIC THRESHOLD ESTIMATES FOR THE EFFECT OF SELECTED POLLUTANTS ON ASTHMA ATTACK RATES[a]**

| Pollutant | Minimum daily temperature, °F | Intercept (attack rate per person) | Estimated effects threshold, $\mu g/m^3$ | Slope | Estimated percent excess risk at specified 24-hour exposures |
|---|---|---|---|---|---|
| Total suspended particulates | 30 to 50 | 0.237 | 56 | 0.000254 | 22% at 260 $\mu g/m^3$ |
| Suspended sulfates | 30 to 50 | 0.240 | 11.9 | 0.000371 | 4% at 35 $\mu g/m^3$ |
| | >50 | 0.245 | 7.3 | 0.005009 | 57% at 35 $\mu g/m^3$ |

[a] Threshold functions could not be calculated for sulfur dioxide or for other temperature ranges involving the tabulated pollutants.

°F), excess asthma attacks were first noted at total suspended particulate levels of 56 $\mu g/m^3$ (Figure 5.4.5). The dose-response function further estimates that elevations of total suspended particulates to the once-yearly allowable level of 260 $\mu g/m^3$ might be expected to induce a 22 percent increase in asthma attack rates. No threshold fit for total suspended particulates could be made for the large number of freezing days or the smaller number of warmer days. Threshold functions could be fitted for suspended particulate sulfates (Figure 5.4.6). Levels of 12 $\mu g/m^3$ induced modest excesses in asthma on cooler days ($T_{min}$ = 30 to 50 °F). More striking excesses at a lower threshold (7.3 $\mu g/m^3$) were found for warmer days ($T_{min} > 50$ °F). The dose-response function predicts that a suspended sulfate exposure of 35 $\mu g/m^3$ would be associated with a 4 percent increase in asthma attacks on cooler days and a 57 percent increase on warmer days.

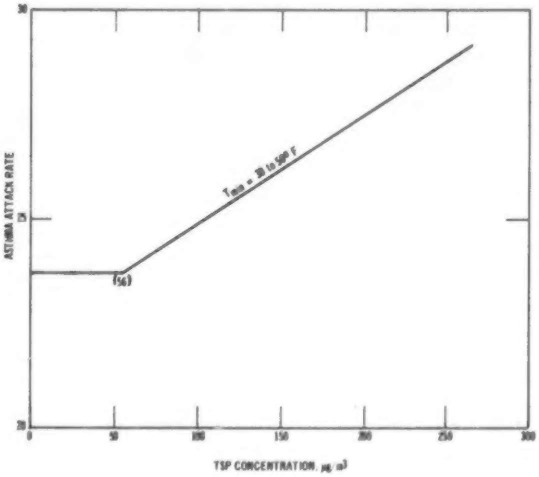

Figure 5.4.5. Effect of total suspended particulates with minimum daily temperature between 30 and 50°F on asthma attack rate.

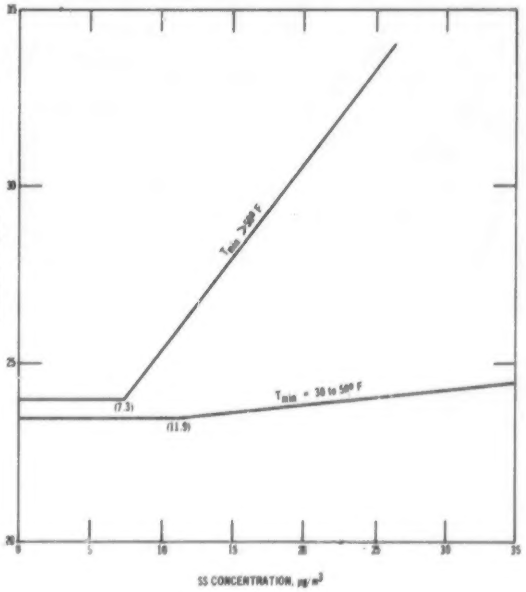

Figure 5.4.6. Effect of minimum daily temperature and suspended sulfates on daily asthma attack rate.

## DISCUSSION

Excessive asthma attack rates attributable to air pollutants occurred only on days when minimum temperatures were above freezing. Both elevated levels of total suspended particulates and suspended sulfates were linked to increases in asthma attacks, and threshold levels were calculated for both. The threshold level for suspended sulfates at cool temperatures ($T_{min}$ = 30 to 50 °F) was higher (12 $\mu g/m^3$) than that found for warmer temperatures (7.3 $\mu g/m^3$ for $T_{min} > 50$ °F). Only a single suspended particulate threshold could be calculated: 56 $\mu g/m^3$ for cooler days. The implications of these thresholds are substantial. Actual monitoring data (1971-1972) showed that suspended particulates in New York City will exceed the calculated threshold in residential areas 50 to 70 percent of all days. The picture for suspended sulfates is equally disturbing: summer exposures in all communities, including the Low exposure area, exceeded the warm weather threshold on 50 to 70 percent of all days, and the threshold for lower temperatures was exceeded on half of fall and winter days.

The findings reported here should be interpreted in light of several possible qualifying factors. Although the study was conducted during the low-pollen season, the possibility that plant allergens may have been present during the few warm weeks at the beginning and at the end of the surveillance period must be considered. Efforts by numerous investigators to isolate and quantify pollen effects have met with varying levels of success. A promising method, measurement of protein content of ambient air, may offer a reliable means in the future for evaluating this variable. In the present study, the net effect of plant allergens would have been to obscure the pollutant associations upon all three asthma panels. Although the study design may have eliminated the pollen season problem for a large part of the surveillance period, it also limited surveillance largely to a period when ambient temperatures were near or below the freezing level. This limitation could account for finding little or no temperature effect, although temperature has been observed to be the strongest determinant of asthma attacks in other studies.[1,9,21,22]

Another problem is the possible role of other unmeasured pollutants, which might have interacted with the pollutants that were measured to produce the observed deleterious health effects. There is, after all, reasonable evidence to speculate that suspended sulfates in the more polluted communities might be combined with fine particulate metals to form more potent irritants than suspended sulfates in the less polluted area, which may be partially of oceanic origin. Higher metal contents were recorded in the more polluted communities for both dustfall and suspended particulate samples.

Other intervening variables of importance may be associated with intrinsic characteristics of the study population. For instance, self-pollution by cigarette smoking might obscure the effects caused by ambient air pollution. Paradoxically, asthmatic smokers seemed to be as responsive to air pollutants as asthmatic nonsmokers. Perhaps the sicker asthmatics, who are most prone to severe repeated attacks, are less likely to smoke and may even be so stressed that they respond somewhat less to air pollutants. Validity of reported asthma attacks is another possible intervening variable. It was not possible in the present study to validate asthmatic episodes by physician visits as was done in previous investigations.[9] However, past validations showed that panelists recognized asthmatic attacks and accurately report them. Thus, there is little reason to believe that clinical findings in the present study would have revealed a study bias.

After weighing the available evidence, we concluded that 24-hour suspended sulfate levels of 12 $\mu g/m^3$ on cooler days ($T_{min}$ = 30 to 50 °F) and 7.3 $\mu g/m^3$ on warmer days ($T_{min} > 50$ °F) were thresholds for the induction of excessive asthma attacks. Total suspended particulate levels of 56 $\mu g/m^3$ on cooler days ($T_{min}$ = 30 to 50 °F) constituted a similar threshold. No firm evidence could be found to associate elevations in sulfur dioxide (100 to 180 $\mu g/m^3$ on 10 percent of days) with excessive asthma attack rates on either cold or warmer days.

## SUMMARY

Increased asthma attack rates among 148 panelists, who completed weekly diary reports, were linked to suspended particulate and suspended sulfate air pollution. Pollutants exerted an effect only on days in which the minimum temperature was above freezing ($T_{min}$ = 30 to

50 °F). The estimated threshold level for total suspended particulates was very low, 56 $\mu g/m^3$, and that for suspended sulfates was 12 $\mu g/m^3$. On warmer days ($T_{min} > 50$ °F), the threshold estimate for suspended sulfates was lower, 7.3 $\mu g/m^3$, and no good estimate for the total suspended particulate threshold could be established. Suspended particulate and suspended sulfate air pollution, like ambient temperature, may very well eventually be identified as important determinants of asthma attack rates in urban America.

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# 5.5 FREQUENCY AND SEVERITY OF CARDIOPULMONARY SYMPTOMS IN ADULT PANELS: 1970-1971 NEW YORK STUDIES

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## INTRODUCTION

Elderly people, particularly those with heart or lung disease, seem vulnerable to very high levels of air pollutants as shown by their increased mortality and morbidity during air pollution episodes.[1-12] However, the influence of smaller day-to-day pollutant fluctuations on the health of people with chronic heart or lung disease is less well defined. In Los Angeles, hospital admission rates for people with bronchitis were shown to increase with increasing daily concentrations of ambient sulfur dioxide.[13] During several winter seasons in London, panels of bronchitics were asked to maintain daily diaries indicating their overall state of health.[14] They reported increased morbidity when ambient concentrations of sulfur dioxide exceeded 600 $\mu g/m^3$. Patients with chronic bronchitis in Chicago were asked to maintain a record of dates of onset and duration of acute respiratory illness, emergency visits to physicians or hospitals, and hospitalizations.[15] Increased acute morbidity among severe bronchitics over age 55 was associated with sulfur dioxide concentrations of 150 to 300 $\mu g/m^3$ or greater.

The present study investigated the influence of both air pollution and meteorologic conditions, as indexed by temperature, upon selected daily symptom rates in elderly subjects. We hypothesized that air pollutants exert a significant influence on cardiopulmonary symptom rates independent of the influence of temperature. Our purposes were to document pollutant thresholds for adverse health effects, to define dose-response relationships between air pollutants and the aggravation of cardiac or respiratory symptoms, and to estimate the relative importance of fluctuations in temperature and fluctuations in pollutant levels.

## METHODS

### Community Selection

Three socioeconomically similar communities were selected on the basis of air quality measurements supplied by the New York City Department of Air Resources (NYC-DAR) and the Suffolk County (New York) Department of Health, current information on house and property values, and the most recently available census data. Riverhead, Long Island, was chosen as a Low exposure community, the Howard Beach section of Queens as an Intermediate exposure community, and the Westchester section of the Bronx as a High exposure community. Due to recent changes in air quality, the Queens and Bronx communities were found to have similar current pollutant concentrations, often below National Primary Air Quality Standards. Hence, the latter two communities were redesignated Intermediate I and Intermediate II, respectively.

### Population Selection

Participants were selected from lists provided by the New York City Housing Authority, local Golden Age clubs, and religious organizations. Each prospective participant was personally interviewed concerning the presence of cough, phlegm production, angina pectoris, enlarged heart, shortness of breath, leg swelling, heart attack history, occupational exposure to pulmonary irritants, personal smoking habits, residential mobility, age, sex, race, and general socioeconomic status. Based upon this interview, participants were classified (according to criteria given elsewhere)[16] as "well" when no significant history of cardiac or respiratory disease was elicited, as having "heart disease" when a history of cardiac symptoms was reported, as having "lung disease" when a history of respiratory symptoms was obtained, and as having "heart and lung disease" when both cardiac and respiratory symptoms were reported. Within each community, between 190 and 340 subjects at least 60 years old were enrolled. About half of the subjects were classified as healthy, with the remainder being fairly evenly distributed over the three disease categories. All subjects lived within 1.5 miles of the air pollution monitoring stations.

### Symptom Reporting

Different diaries were distributed to each of the four panels, as detailed elsewhere.[16] Where common symptoms could be expected, similar questions were asked. On a daily basis, "well" panelists were asked about the presence of chest pains, wheezing, cough, and phlegm; "heart" panelists were asked about shortness of breath, angina or dull chest pain, and leg swelling; "lung" panelists were asked about cough or phlegm, wheezing and shortness of breath, angina or dull chest pain, leg swelling, and cough or phlegm. Participants were asked to indicate for each day whether their symptoms were never present, better, the same, worse, or much worse than usual. On a weekly basis, all panelists were asked about the presence of a cough, cold, or sore throat, a visit to a doctor due to illness, or an admission to the hospital. All panelists were asked to maintain their symptom diaries daily for the 32-week period October 8, 1970, through May 22, 1971.

### Pollution and Weather Monitoring

One pollution monitoring station was established in each community to provide a representative sampling of population exposure. At each station, sulfur dioxide ($SO_2$), total suspended particulates (TSP), suspended sulfates (SS), suspended nitrates (SN), and nitrogen dioxide ($NO_2$) were monitored continuously and analyzed on a 24-hour sample basis.[17] Dustfall was also monitored continuously, but analyzed on a monthly sample basis. Continuous measurements of temperature, humidity, barometric pressure, and wind speed and direction were obtained from nearby airports. A more detailed description of the monitoring program is presented elsewhere.[17]

### Statistical Analyses

Specific symptom rates were computed for each of the 224 days. The numerator contained a count of all people who responded "worse" or "much worse." This pooling was necessary since there were very few "much worse" responses. The denominator contained a count of all people who answered "much worse," "worse," "same," "better," or "never present." The rate is, in effect, a measure of the percent of people at risk who felt worse on a given day. A more general index was based upon whether or not panelists reported aggravation of any existing cardiopulmonary symptom or induction of any new symptom. This index was designated the "combined symptom" variable.

The statistical analyses involved six steps. First, intrinsic differences between panels and communities were evaluated. All additional steps considered temporal rather than spatial variations. Second, seasonal patterns were sought by plotting intracommunity average weekly symptom attack rates and weekly averages for each pollutant. Third, simple correlations between symptom rates and pollutant levels were calculated for each community and possible interrelationships were defined. Fourth, stepwise multiple regression analysis sought to identify pollutant effects after removal of any linear temperature effects.[18] Fifth, temperature-specific relative risk models for the induction or aggravation of symptoms were constructed for selected pollutant levels within each community, and a combined estimate of pollutant effects on the total elderly population was made. Sixth, temperature-specific estimates of linear or threshold functions for pollutants were made, and the impact was estimated for existing air quality standards and current air pollution levels.[19,20]

## RESULTS

### Pollutant Exposure

Short-term peak exposures to air pollutants, rather than long-term exposures, would most likely be significant determinants of day-to-day variations in the severity of cardiopulmonary symptoms. Consequently, median, 90th percentile, and maximum pollutant levels were tabulated for each study community and for each season (Table 5.5.1).

Aerometric data collected during the study period substantiated the community difference indicated by long-term pollution levels[17] between the Low pollution community and the two Intermediate communities. During the study (fall to spring in Table 5.5.1), median concentrations of sulfur dioxide (13 to 18 $\mu g/m^3$) and total suspended particulates (29 to 32 $\mu g/m^3$) in the Low exposure community were about half the levels of the Intermediate communities. The differences for suspended nitrates and suspended sulfates were smaller, with nitrate levels of 1 to 3 $\mu g/m^3$ and sulfate levels of 6 to 14 $\mu g/m^3$ being recorded for the three communities. The difference in long-term annual average exposures between the two Intermediate communities was slight for all of the pollutants previously mentioned.

As expected from study of longer term averages, the Intermediate exposure communities almost always exhibited higher concentrations at all three points on the cumulative frequency distribution. Intermediate communities had much

### Table 5.5.1. DAILY AIR POLLUTANT EXPOSURE PATTERNS IN THREE NEW YORK COMMUNITIES DISTRIBUTED BY SEASON

| Pollutant and community | Daily pollutant levels, μg/m³ | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Median | | | | 90th percentile | | | | Maximum | | | |
| | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 | Fall 1970 | Winter 1971 | Spring 1971 | Summer 1971 |
| Sulfur dioxide | | | | | | | | | | | | |
| Low | 13 | 18 | 15 | 8 | 45 | 76 | 45 | 21 | 131 | 125 | 114 | 52 |
| Intermediate I | 56 | 34 | 48 | 29 | 120 | 119 | 108 | 71 | 231 | 287 | 143 | 107 |
| Intermediate II | 64 | 35 | 19 | 21 | 178 | 162 | 104 | 58 | 268 | 349 | 219 | 123 |
| Total suspended particulates | | | | | | | | | | | | |
| Low | 32 | 29 | 29 | 34 | 59 | 56 | 53 | 58 | 97 | 134 | 91 | 104 |
| Intermediate I | 56 | 55 | 57 | 60 | 93 | 90 | 102 | 101 | 170 | 156 | 193 | 152 |
| Intermediate II | 57 | 75 | 77 | 78 | 118 | 121 | 128 | 118 | 216 | 188 | 217 | 211 |
| Suspended sulfates | | | | | | | | | | | | |
| Low | 11 | 9 | 6 | 9 | 23 | 18 | 16 | 22 | 48 | 51 | 32 | 42 |
| Intermediate I | 14 | 12 | 9 | 12 | 22 | 20 | 17 | 27 | 28 | 39 | 36 | 37 |
| Intermediate II | 14 | 14 | 10 | 14 | 22 | 23 | 22 | 32 | 36 | 35 | 42 | 53 |
| Suspended nitrates | | | | | | | | | | | | |
| Low | 1 | 1 | 1 | 2 | 3 | 2 | 2 | 4 | 3 | 6 | 4 | 6 |
| Intermediate I | 2 | 1 | 2 | 4 | 3 | 3 | 5 | 8 | 6 | 6 | 11 | 15 |
| Intermediate II | 1 | 2 | 3 | 4 | 3 | 4 | 9 | 9 | 5 | 6 | 20 | 14 |

higher peak levels of sulfur dioxide, total suspended particulates, and suspended nitrates. Somewhat surprisingly, intercommunity differences in suspended sulfates were quite small. Levels in the Low exposure community were higher than expected, possibly reflecting penetration by sulfates from the more urbanized communities, or even sulfates of oceanic origin. More predictably, dustfall metals and respirable particulate levels were higher in the Intermediate communities. None of the study communities reported sulfur dioxide concentrations that exceeded the National Daily Primary Air Quality Standard (365 μg/m³). Short-term total suspended particulate measurements were also within the allowable standard (260 μg/m³).

Nitrogen dioxide concentrations were calculated and were included in the statistical analyses. These analyses indicated that nitrogen dioxide levels in the three communities were relatively low. However, recent reevaluation by the Environmental Protection Agency indicates that the measurement method for nitrogen dioxide is subject to interferences and variable collection efficiencies. Therefore, these data, while summarized elsewhere,[17] are not included in this report.

In summary, all three communities achieved both short- and long-term air quality standards for sulfur dioxide. The two Intermediate communities just exceeded the allowable annual average level of total suspended particulates but did not violate the short-term standard. The Low exposure community had total suspended particulate levels well below allowable levels for either short- or long-term exposure. Clearly, any excess morbidity attributable to short-term exposures involving total suspended particulates or sulfur dioxide occurred despite the fact that these communities were within currently allowable exposure limits.

## Characteristics of the Study Population

Characteristics of the study population are given in the Appendix, Table 5.5.A.1. Panelists in the Low pollution community were younger than those in the Intermediate communities; i.e., a greater proportion of panelists were less than 70 years old. Based upon background symptom histories obtained by personal interview, there were no major differences among the "well" panelists. "Heart" as well as "lung" panelists in the Low pollution community were somewhat less symptomatic than those in the Intermediate communities, with the only major difference occurring for the symptom shortness of breath, for which panelists with both "heart and lung" disease reported the highest background. Otherwise, panelists with both "heart and lung" disease reported the highest background symptom rates of all groups, but no large differences were observed between the three communities.

A marked decrease in panelist participation occurred over the course of the year in spite of frequent phone calls to remind those people who did not return their diaries on time (Table 5.5.A.2). Over the course of the study, three of four panels in each of the Intermediate pollution communities had a dropout rate exceeding 50 percent of those initially enrolled. The lowest attrition rates were observed in the Low pollution community, where all panels retained at least 70 percent of enrollees. At the end of the study, there were 142, 117, and 144 active panelists in the Low, Intermediate I, and Intermediate II communities, respectively.

To gain a better idea of gross intercommunity differences in symptom reporting, mean daily symptom rates were determined for each panel within each community (Table 5.5.2). As expected, intra- and intercommunity differences were apparent. "Well" panelists usually had the lowest symptom rates. "Heart and lung" panelists had the highest reported symptom rates, and the rates of the other two panels were intermediate. For all panels and all symptom reporting, rates were lowest in the Low pollution community. As previously indicated, the baseline demographic characteristics and symptom status of various panels were not the same in each community (Table 5.5.A.1). Differences in daily symptom rates between communities, shown in Table 5.5.2, should be attributed to the fact that panels in Intermediate neighborhoods were older, had more smokers, and were otherwise inherently different from the Low exposure panel at the start of the study. In the Intermediate communities, panelists generally reported symptom rates 2 to 5 times those of the Low pollution community. The symptom ratios for shortness of breath and angina among "heart" panelists in the Intermediate communities were even greater, being 8 to 13 times those reported in the Low pollution community. No major differences in symptom rates were observed between the Intermediate communities. Fortunately, differences between panelists in the Intermediate communities were small, thus allowing

Table 5.5.2. MEAN DAILY SYMPTOM RATES OF PANELISTS WITH RESPECT TO THE LOW POLLUTION COMMUNITY

| Panel | Symptom | Low pollution community symptom rate[a] | Intercommunity symptom ratios (Intermediate/Low) | |
|---|---|---|---|---|
| | | | Intermediate I | Intermediate II |
| Well | Cough and phlegm | 7.0 | 2.2 | 2.3 |
| | Chest pain | 0.8 | 4.1 | 4.5 |
| | Wheezing | 1.1 | 2.6 | 4.5 |
| Heart | Shortness of breath | 1.6 | 12.8 | 11.6 |
| | Angina or chest pain | 4.2 | 7.9 | 8.6 |
| | Leg swelling | 2.2 | 5.4 | 2.4 |
| Lung | Shortness of breath | 5.2 | 4.6 | 3.6 |
| | Cough and phlegm | 10.3 | 2.2 | 2.0 |
| | Wheezing | 4.9 | 3.9 | 3.0 |
| Heart and lung | Shortness of breath | 6.7 | 3.7 | 4.4 |
| | Cough and phlegm | 11.9 | 1.9 | 2.9 |
| | Angina or chest pain | 9.1 | 2.9 | 4.2 |
| | Leg swelling | 2.2 | 6.1 | 8.2 |

[a] Mean daily rate is based upon the number of respondents marking worse or much worse x 100 divided by the total number of panelists responding to that day's diary question.

the pooling of panels if this became necessary. We concluded that there were gross intercommunity differences that reflected a combination of intrinsic intercommunity differences in the symptom severity and possibly an additional component attributable to fluctuations in ambient temperature and pollutant levels. Since the study was not designed to compare intercommunity response rates, further analyses were restricted to temporal variations in symptom rates within each community, thus essentially eliminating the problem of intrinsic intercommunity difference.

## Temporal Patterns

Insight into the major determinants of the aggravation of cardiopulmonary symptoms might be gained by evaluating seasonal trends in symptom reporting, ambient temperature, and ambient pollutant level. Once identified, these determinants could be isolated or adjusted for in later analyses. For this reason, weekly averages were compiled for the "combined symptom" rate in each panel and plotted over the weeks of study. Similar plots were prepared for temperature and for each pollutant. Worsening of any one of the symptoms that comprised the combined cardiopulmonary index proved the most stable indicator. For illustrative purposes, the experience of panelists with combined "heart and lung" disease living in the Low and Intermediate I exposure areas is presented in Figure 5.5.1. Similar plots were evaluated for all panels in every community. Few consistent seasonal trends were noted among panels or within communities. There was a slight tendency towards increased symptom reporting during late fall and winter among three of the four panels: the "well," "lung," and "heart and lung" categories. Since fluctuations in temperature are accompanied by fluctuations in mortality caused by heart and lung disease in the elderly population,[21] it seemed reasonable that ambient temperature might be an important determinant of symptom severity in elderly patients. Moreover, low temperatures have been shown to aggravate asthma[22] and might therefore aggravate symptoms caused by other chronic pulmonary disorders. In the two Intermediate communities, "heart" panelists reported higher rates during the earlier fall weeks of the study, after which the rates gradually subsided.

Exceptions to these trends were not infrequent, as illustrated in the Intermediate I community (Figure 5.5.1) by the percent of panelists with "heart and lung" disease who reported a worsening of symptoms that manifested absolutely no seasonal trend. One other possibly important observation should be stressed: there was no obvious overreporting in the early weeks of the study.

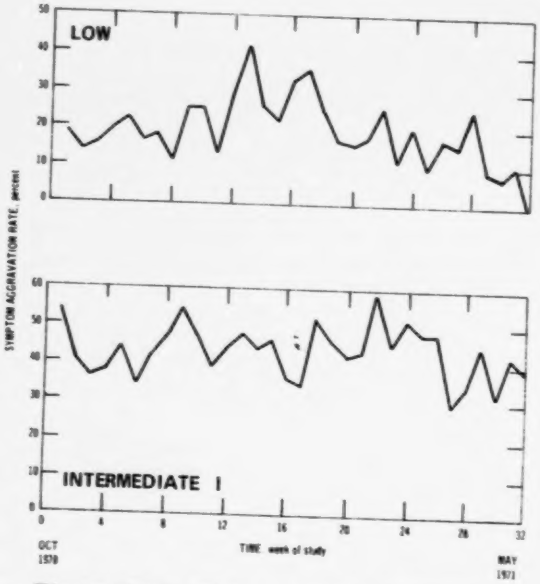

Figure 5.5.1. Weekly symptom aggravation rates, "heart and lung" panel.

As expected, pollutant levels followed seasonal trends that were actually summations of the processes that govern pollutant emissions, i.e., atmospheric transformation and atmospheric dispersion. Seasonal trends in environmental factors were sequentially reviewed. Ambient temperature followed a gently U-shaped curve, with temperatures falling from the beginning of the study until late February (week 17) (Figure 5.5.2). Sulfur dioxide levels rose as ambient temperature fell, with the Intermediate communities attaining daily levels twice those of the Low exposure community (Figure 5.5.3). Simultaneous plots of symptom aggravation rates and exposure suggested a temporal correlation between symptom ratio and sulfur dioxide in the Low exposure community. Sulfur dioxide levels were low during March and early April, perhaps because dispersing processes were more than adequate even though emissions related to combustion of fossil fuels for heating might still have been substantial. On the other hand, total suspended particulate levels did not exhibit any

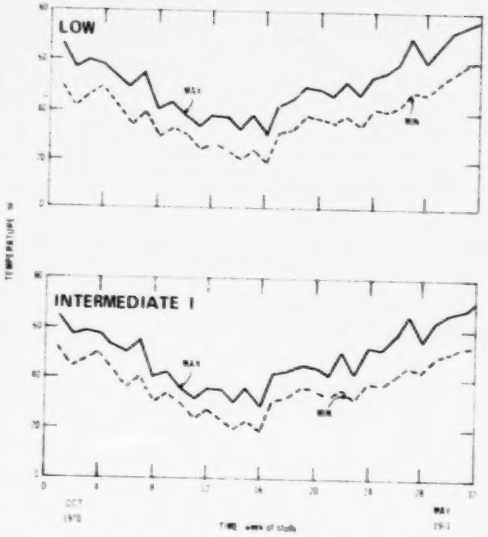

Figure 5.5.2. Weekly average maximum and minimum temperatures during study (1970-1971).

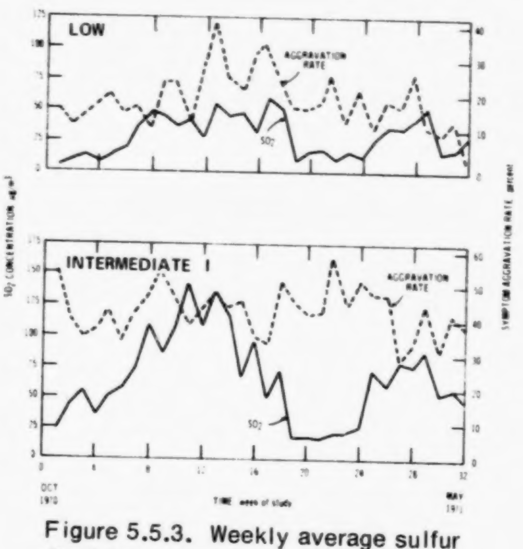

Figure 5.5.3. Weekly average sulfur dioxide concentrations and symptom aggravation rates for "heart and lung" panel.

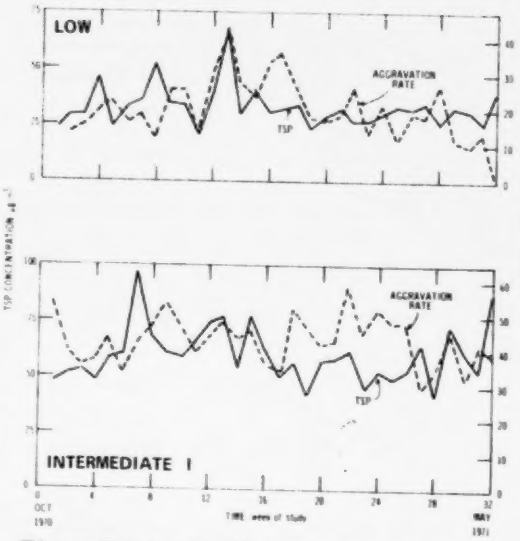

Figure 5.5.4. Weekly average total suspended particulate concentrations and symptom aggravation rates for "heart and lung" panel.

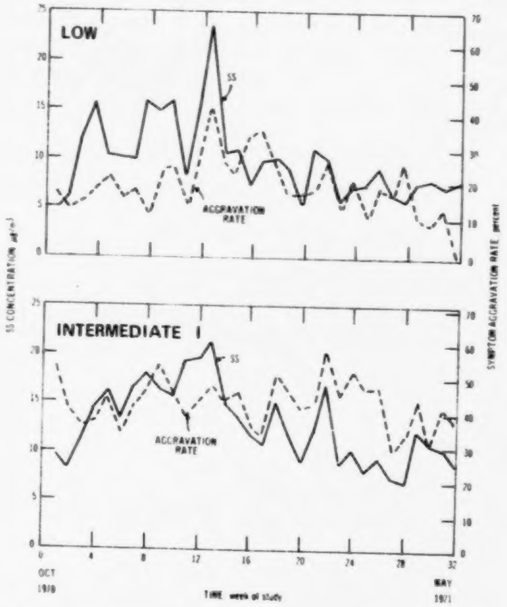

Figure 5.5.5. Weekly average suspended sulfate concentrations and symptom aggravation rates for "heart and lung" panel.

clear seasonal trends (Figure 5.5.4). Levels were somewhat lower in the first month of the study and seemed related to symptom rates in the Low but not the Intermediate communities. Like sulfur dioxide, the sulfate fraction of total suspended particulates showed a clear seasonal trend in the more polluted communities (Figure 5.5.5);

even the Low exposure community was found to have large variations in wintertime suspended sulfate levels, with some peak weekly averages higher than those found in more polluted communities. The simultaneous symptom rate plot suggested that sulfates were an important determinant. The nitrate fraction of total suspended particulates followed somewhat different temporal patterns in the Low and Intermediate exposure communities, but no consistent seasonal trend was discerned (Figure 5.5.6). The temporal pattern of nitrates could not be easily related to simultaneous plots of symptom rates.

An overview of seasonal trends leads to the following observations: (1) The effects of temperature and pollutants were likely to be entangled in a complex fashion. (2) The effect of temperature could well mask effects attributable to pollutants, especially any sulfur dioxide effects. (3) Suspended sulfates might well constrain or entirely preclude any statistical analyses that utilized the Low exposure community as a control population. On the other hand, intrusion of relatively high suspended sulfate concentrations into the Low exposure community provided a rare double-blind study of air pollution effects since neither panelists nor survey teams could have any idea that the Low exposure community was in fact quite polluted.

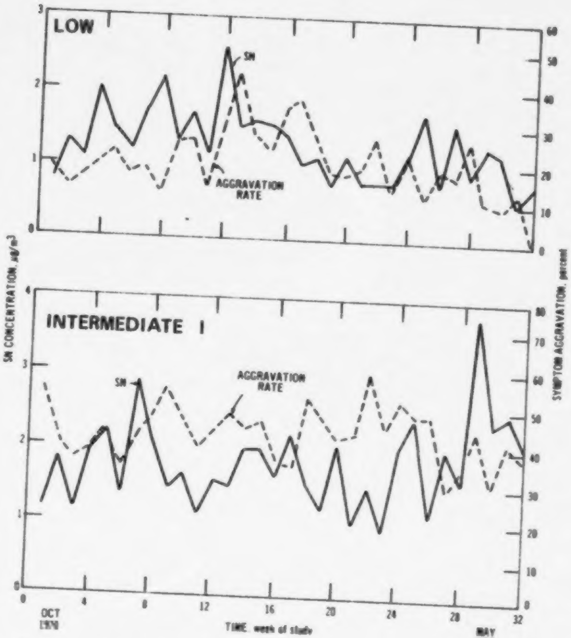

Figure 5.5.6. Weekly average suspended nitrate concentrations and symptom aggravation rates for "heart and lung" panel.

**Table 5.5.3. NUMBER OF STATISTICALLY SIGNIFICANT CORRELATIONS BETWEEN VARIOUS SYMPTOMS AND DAILY POLLUTANT OR TEMPERATURE LEVELS**

| Symptom | Direction of correlative | Number of correlations | | | | | |
|---|---|---|---|---|---|---|---|
| | | TSP | SN | SS | $SO_2$ | Max. temp | Min temp |
| Shortness of breath | Positive | 1 | 0 | 6 | 1 | 1 | 2 |
| | Negative | 0 | 1 | 0 | 2 | 4 | 3 |
| Cough and phlegm | Positive | 2 | 1 | 6 | 4 | 1 | 1 |
| | Negative | 0 | 3 | 0 | 2 | 8 | 7 |
| Angina or chest pain | Positive | 1 | 0 | 5 | 5 | 2 | 3 |
| | Negative | 0 | 0 | 0 | 2 | 3 | 2 |
| Wheezing | Positive | 1 | 1 | 3 | 2 | 0 | 0 |
| | Negative | 1 | 1 | 0 | 1 | 4 | 3 |
| Leg swelling | Positive | 1 | 0 | 1 | 0 | 1 | 4 |
| | Negative | 1 | 0 | 0 | 2 | 0 | 0 |

### Simple Correlation Analyses

In attempting to define effects thresholds or dose-response functions for pollutants, a hierarchical approach seemed advisable. Consequently, we first evaluated community-specific simple correlations for individual symptoms and for the combined symptom index within each panel before proceeding to multiple regression analyses (Tables 5.5.3, 5.5.4, and 5.5.A.3). The individual symptom rates and combined symptom index were often strongly and negatively correlated with temperature. Among the "heart" panelists, however, symptoms were positively correlated with temperature. Suspended sulfate and sulfur dioxide were the only pollution variables that significantly correlated with the combined symptom rate in a consistently positive manner. Fewer significant correlations with symptom rates were observed for total suspended particulates and suspended nitrates.

When the symptoms shortness of breath, cough and phlegm, angina or chest pain, wheezing, and leg swelling were considered in relation to daily variations in air quality. The strongest and most consistent positive correlations with suspended sulfate pollution were observed for shortness of breath, cough and phlegm, and angina or chest pain. Additionally, the symptoms

Table 5.5.4. SIMPLE CORRELATION OF SYMPTOM AGGRAVATION WITH POLLUTANTS AND AMBIENT TEMPERATURE[a]

| Panel | Community | Correlation coefficient | | | | |
|---|---|---|---|---|---|---|
| | | $SO_2$ | TSP | SS | SN | Max. temp |
| Heart | Low | -0.097 | -0.131 | -0.070 | -0.042 | 0.039 |
| | Intermediate I | -0.005 | 0.032 | 0.172[b] | 0.018 | 0.047 |
| | Intermediate II | 0.091 | -0.107 | 0.086 | -0.133 | -0.120 |
| Lung | Low | 0.010 | 0.005 | 0.015 | 0.146[b] | -0.205[c] |
| | Intermediate I | 0.143[b] | 0.053 | -0.356[c] | -0.155[b] | -0.511[c] |
| | Intermediate II | -0.291[c] | 0.054 | 0.047 | 0.103 | 0.097 |
| Heart and lung | Low | 0.230[c] | 0.160[b] | 0.282[c] | 0.158[b] | -0.331[c] |
| | Intermediate I | -0.088 | -0.053 | 0.264[c] | -0.087 | -0.145[b] |
| | Intermediate II | 0.078 | 0.088 | 0.178[b] | -0.019 | -0.191[c] |
| Well | Low | 0.140[b] | 0.009 | 0.021 | -0.003 | -0.247[c] |
| | Intermediate I | 0.008 | -0.055 | 0.199[c] | -0.173[b] | -0.399[c] |
| | Intermediate II | 0.206[c] | 0.106 | 0.204[b] | 0.049 | -0.411[c] |

[a] Symptom aggravation for lung panelist involved worsening of dyspnea, cough, or phlegm production; for heart panelists, increased frequency or severity of angina or dyspnea; for patients with both disorders, worsening of any symptom. For well panels, appearance of cough, phlegm, or angina was regarded as aggravation of a chronic disorder.

[b] $p < 0.05$.

[c] $p < 0.01$.

angina and cough and phlegm were frequently positively correlated with sulfur dioxide, although some inconsistencies existed. Wheezing and leg swelling showed no consistent pattern relating to air pollution.

When the four panels were considered as health indicators of daily variations in air pollution, the "heart and lung" panelists proved the most reliable index (Table 5.5.4). Among the "heart and lung" panelists, significant positive correlations with suspended sulfates were found in all communities for the symptoms shortness of breath, cough and phlegm, and angina. Among the other three panels, significant associations of symptom rates with pollution were fewer, and the Intermediate I community was seemingly responsible for most of the significant associations between symptom rates and suspended sulfates. Based upon the principal relationships suggested by the simple correlations, further analysis focused primarily on the combined symptom index and on the three symptom categories cough and phlegm, dyspnea, and angina.

It was also necessary to review the relationships linking pollutants to each other and to ambient temperature and this was done by study of a simple correlation matrix (Table 5.5.5). Every pollutant in every community was highly correlated with total suspended particulates. Likewise, every pollutant except total suspended particulates was correlated significantly with ambient temperature as indexed by either maximum ($T_{max}$) or minimum ($T_{min}$) daily temperatures. As expected from their seasonal trends, sulfur dioxide and suspended sulfates increased as ambient temperature fell, and suspended nitrates decreased as temperature fell. Another important relationship was the strong positive association between sulfur dioxide and suspended sulfates. In summary, study of the simple correlation matrix served three purposes. First, attention was focused upon three specific symptom categories and a combined symptom index; second, strong associations between symptom rates and temperature demonstrated the need to isolate this variable in subsequent analyses; and third, complex relationships between pollutants and temperature were demonstrated.

Table 5.5.5. SIMPLE CORRELATIONS OF POLLUTION AND TEMPERATURE

| Pollutant | Community | Correlation coefficient | | | |
|---|---|---|---|---|---|
| | | $SO_2$ | SS | SN | Max. temp |
| TSP | Low | 0.364[a] | 0.795[a] | 0.483[a] | 0.006 |
| | Intermediate I | 0.381[a] | 0.506[a] | 0.402[a] | 0.029 |
| | Intermediate II | 0.230[a] | 0.625[a] | 0.604[a] | 0.075 |
| $SO_2$ | Low | | 0.369[a] | 0.118 | -0.266[a] |
| | Intermediate I | | 0.339[a] | 0.076 | -0.195[a] |
| | Intermediate II | | 0.388[a] | -0.026 | -0.365[a] |
| SS | Low | | | 0.356[a] | -0.153[b] |
| | Intermediate I | | | 0.112 | -0.255[a] |
| | Intermediate II | | | 0.289[a] | -0.241[a] |
| SN | Low | | | | -0.078 |
| | Intermediate I | | | | 0.222[a] |
| | Intermediate II | | | | 0.258[a] |

[a] $p < 0.01$.

[b] $p < 0.05$.

## Stepwise Multiple Regression Analyses

Since symptoms in most panelists seemed strongly influenced by ambient temperature levels, multiple regression techniques were employed to remove all symptom variation that would be attributed to temperature before considering the effect of any pollutant. As previously discussed, several important pollutants, including sulfur dioxide and suspended sulfates, were inversely associated with ambient temperature. After removal of temperature effects, the effect of each single pollutant was considered. Next, pollutants were evaluated in combination until each possible permutation was considered. Any effects on symptoms due to these pollutants could easily be obscured by the planned multiple regression. Thus, multiple regression estimates of the effects of these pollutants were quite conservative. A second qualification should be made before interpreting the multiple regression analysis: the effects of temperature upon symptom rates are not really simple or linear. Symptoms tend to be aggrated by extremely low and by excessively high ambient temperatures. Thus, multiple regression might cause spurious associations between pollutants such as sulfur dioxide and suspended sulfates that decrease with elevations in temperature. Nevertheless, multiple regression could prove useful in selecting pollutants for temperature-specific analyses and in interpreting such analyses.

A probability matrix was employed to summarize the multiple regression analyses performed for the combined symptom variable and for the three specific symptoms thought most likely to be responsive to fluctuations in air pollutants (Table 5.5.6). The different panels did not respond in a homogeneous fashion. Because of the infrequent reporting of any single symptom, only the combined symptom variable could be analyzed for the "well" panels. The "well" and "heart disease" panels also showed less frequent significant effects than the other panels. The sickest panel, those with "heart and lung" disease, reported significant symptom aggravations attributable to environmental factors most frequently. The "lung disease" panel followed a pattern quite similar to panelists with "heart and lung" disease.

As indicated by the preliminary analyses, ambient temperature was the environmental factor that most frequently exerted a significant influence on the cardiopulmonary symptom status of every panel. Decreasing ambient temperatures were usually accompanied by symptom aggravation and increased symptom reporting. In the "heart" panel, temperature was related to an opposite effect. The Low exposure community more consistently reported significant temperature effects than the two Intermediate communities.

All of the air pollutants were linked to significant fluctuations in cardiopulmonary symptoms in one or more panels and in one or more communities. The strongest and most consistent pollutant effects were found for suspended sulfates, which were linked to a worsening of symptoms. Inclusion of the sulfate variable just after temperature left no significant increases in symptom aggravation that were attributable to other pollutants. On the other hand, the worsening of symptoms attributable to suspended sulfates always persisted after the removal of effects attributable to temperature and all other pollutants. In no case was an increase in suspended sulfates coupled with an amelioration of symptoms. As previously discussed, the significant aggravating effect of sulfates is especially noteworthy because multiple regression analyses could tend to wash out any sulfate effect. The other two pollutants that seemed most likely to be affecting panelists adversely were suspended particulates and sulfur dioxide. Suspended particulate levels, which were largely independent of temperature, seemed to predict symptom aggravation concurrent with, but not nearly as well as, suspended sulfates. Sulfur dioxide effects, which like suspended sulfate effects were clouded by the initial removal of all effects associated with temperature, were most frequently related to symptom aggravation in the "heart and lung" panel. In the "heart" and "lung" panels, elevations in sulfur dioxide were at times accompanied by a beneficial effect, probably spurious, that could result either from chance alone or from the previously recounted, probably nonlinear, temperature effect.

Suspended nitrate concentrations exerted scattered significant effects that appeared inconsistent in direction. Although the Low exposure community was involved in more than half of

## Table 5.5.6. SUMMARY OF MULTIPLE REGRESSION ANALYSES FOR EFFECTS OF TEMPERATURE AND POLLUTANTS ON SYMPTOM AGGRAVATION

| Panel | Symptom | Community | Source of variation[a] | | | | |
|---|---|---|---|---|---|---|---|
| | | | Temperature alone | Temperature-adjusted | | | |
| | | | | $SO_2$ | TSP | SS | SN |
| Well | Combined cardio-pulmonary symptoms | Low | <0.001[b] | NS | NS | NS | NS |
| | | Intermediate I | <0.001[b] | NS | NS | NS | NS |
| | | Intermediate II | <0.001[b] | NS | <0.05 | NS | <0.025 |
| Heart | Combined cardio-respiratory symptoms | Low | NS | NS | NS | NS | NS |
| | | Intermediate I | NS | NS | NS | <0.025 | NS |
| | | Intermediate II | NS | NS | NS | NS | NS |
| | Shortness of breath | Low | NS | NS | NS | NS | <0.10[b] |
| | | Intermediate I | NS | <0.05[b] | NS | NS | NS |
| | | Intermediate II | NS | NS | <0.05 | <0.001 | NS |
| | Angina | Low | <0.005 | <0.05[b] | NS | NS | NS |
| | | Intermediate I | NS | <0.10 | NS | <0.01 | NS |
| | | Intermediate II | <0.10 | NS | NS | NS | NS |
| Lung | Combined cardio-respiratory symptoms | Low | <0.01[b] | NS | NS | NS | <0.10 |
| | | Intermediate I | <0.001[b] | NS | NS | <0.001 | NS |
| | | Intermediate II | NS | <0.001[b] | NS | NS | NS |
| | Shortness of breath | Low | <0.01[b] | NS | NS | NS | NS |
| | | Intermediate I | <0.001[b] | NS | NS | <0.001 | NS |
| | | Intermediate II | NS | <0.005 | NS | NS | NS |
| | Cough and phlegm | Low | <0.005[b] | NS | NS | NS | NS |
| | | Intermediate I | <0.001[b] | NS | NS | <0.005 | NS |
| | | Intermediate II | <0.001[b] | <0.01[b] | NS | NS | NS |
| Heart and lung | Combined cardio-respiratory symptoms | Low | <0.001[b] | <0.025 | <0.025 | <0.001 | <0.10 |
| | | Intermediate I | <0.10[b] | NS | NS | <0.005 | NS |
| | | Intermediate II | <0.01[b] | NS | NS | <0.10 | NS |
| | Shortness of breath | Low | <0.01[b] | NS | <0.025 | <0.001 | <0.05 |
| | | Intermediate I | <0.05[b] | NS | <0.05 | <0.001 | NS |
| | | Intermediate II | NS | NS | NS | NS | NS |
| | Cough and phlegm | Low | NS | NS | NS | <0.025 | NS |
| | | Intermediate I | NS | <0.001 | NS | NS | NS |
| | | Intermediate II | <0.001[b] | NS | <0.10 | <0.05 | NS |
| | Angina | Low | <0.001[b] | NS | NS | NS | NS |
| | | Intermediate I | NS | NS | NS | NS | NS |
| | | Intermediate II | NS | <0.005 | <0.10 | <0.025 | NS |

[a] NS--not significant, $p > 0.10$.

[b] Negative relationship.

the "significant" associations, it is unlikely that the low levels of suspended nitrates found in this community actually aggravated illness. Nitrates were curiously correlated with both increasing temperature and increasing levels of sulfates and sulfur dioxide, even though these latter pollutants were inversely correlated with temperature. Perhaps these two trends resulted in spurious associations for suspended nitrates. Other explanations are also possible.

Multiple regression analyses gave unequivocal evidence that suspended sulfates were closely linked to aggravation of cardiopulmonary symptoms in the sickest elderly population. Weaker, but probably adverse, effects might be attributed to total suspended particulates and sulfur dioxide. Thus, temperature-specific symptom rate analyses could focus on these three pollutants, although it was unlikely that their effects could be completely separated from one another.

## Temperature-specific Relative Risk and Excess Risk Models

Multiple regression analyses probably gave conservative estimates for the effects of sulfur dioxide and suspended sulfates since part of the effects that might be related to these pollutants was removed by the stronger simultaneously associated effect of temperature. Temperature-specific relative risk models avoid the assumption of linear temperature effects and instead consider days within specific temperature ranges. The effects of ambient temperature are not entirely removed since any specific temperature range may be quite broad. Thus, effects attributed to either sulfur dioxide or suspended sulfates might still contain a residual temperature-related aggravating effect. Therefore, temperature-specific analyses that attribute increased relative risks to any one pollutant are liable to somewhat overstate the effect of that pollutant because of enhancement caused by other pollutants and by concurrent small fluctuations in ambient temperature.

Preliminary analyses indicated that the experience of specific panels might be pooled for the two Intermediate areas. Conversely, panels from the Low exposure area were either intrinsically less symptomatic or reported fewer symptoms because of lower pollution levels. In either case, prudence dictated that panels from the Low exposure community be separately considered. The study was partitioned into colder days, when the minimum daily temperature ($T_{min}$) was 20 to 40 °F, and a second category of warmer days, when minimum temperatures were >40 °F. Relative risks were calculated for each temperature-specific category using as a base the symptom aggravation rates for low pollution days. Relative risks were then computed by using the symptom rate for each successively higher pollutant concentration as the numerator and the base rate from the lowest pollution level as the denominator. In the Intermediate communities where pooling was allowable, the relative risks for each community were weighted by the number of person-days at risk that each represented and combined across the two communities to achieve a single temperature-specific relative risk for a given pollutant level. Relative risks based upon fewer than 500 person-days of observation have been identified in the tables since they were quite unstable. Relative risks for each panel were combined into a model, derived from Health Interview Survey estimates of chronic, activity-limiting disorders in the population 65 or older,[23] to show the experience of each panel. The formula used was:

$$\begin{aligned} \text{total} &= 0.683 \text{ well} + 0.219 \text{ heart} \\ &+ 0.080 \text{ lung} + 0.018 \text{ heart and lung} \end{aligned}$$

The temperature-specific relative risk model for sulfur dioxide showed some evidence for increased symptoms at 24-hour exposures greater than 81 $\mu g/m^3$, but many relative risks were unstable because of the relatively few person-days at risk upon which the estimates were based (Table 5.5.7). In general, relative risks showed a tendency to increase on colder days ($T_{min}$ = 20 to 40 °F) and with elevated levels of sulfur dioxide. Only the "heart" panel failed to show some evidence of a sulfur dioxide effect at lower temperatures. Estimates for the elderly population as a whole reflect the heavy weight attached to the "well" panel in the preceding formula. On warmer days ($T_{min}$ > 40 °F) there was less evidence of a sulfur dioxide effect. The more stable pooled rates for the Intermediate communities showed no increase from any panel.

When temperature-specific relative risk rates for total suspended particulate exposures were evaluated (Table 5.5.8), the observed effect was quite small and seemed to involve "lung" and "heart and lung" panelists on the colder ($T_{min}$ = 20 to 40 °F) days. There was also a suggestion

## Table 5.5.7. RELATIVE RISK OF CARDIOPULMONARY SYMTPOM AGGRAVATION IN ELDERLY PANELISTS EXPOSED TO VARYING LEVELS OF SULFUR DIOXIDE[a]

| Panel | $SO_2$ concentration (24 hr), $\mu g/m^3$ | $T_{min}$ = 20 to 40°F | | $T_{min}$ > 40°F | |
|---|---|---|---|---|---|
| | | Low | Pooled Intermediate | Low | Pooled Intermediate |
| Well | ≤60 | 1.00 (1.8) | 1.00 (17.6) | 1.00 (5.9) | 1.00 (14.7) |
| | 61 to 80 | 1.01 | 0.93 | 1.02[b] | 0.89 |
| | 81 to 365 | 1.45 | 1.09 | 0.85[b] | 0.99 |
| Heart | ≤60 | 1.00 (5.7) | 1.00 (38.2) | 1.00 (4.9) | 1.00 (39.4) |
| | 61 to 80 | 0.63[b] | 1.05 | 0.65[b] | 0.98 |
| | 81 to 365 | 0.81[b] | 1.04 | 2.04[b] | 0.90 |
| Lung | ≤60 | 1.00 (17.0) | 1.00 (29.4) | 1.00 (12.9) | 1.00 (24.2) |
| | 61 to 80 | 1.18[b] | 0.90[b] | 1.12[b] | 0.95 |
| | 81 to 365 | 1.01[b] | 0.98 | 0.78[b] | 0.84 |
| Heart and lung | ≤60 | 1.00 (21.4) | 1.00 (52.6) | 1.00 (15.4) | 1.00 (49.4) |
| | 61 to 80 | 1.61[b] | 1.05 | 1.19[b] | 0.86 |
| | 81 to 365 | 1.29[b] | 1.01 | 0.94[b] | 0.93 |
| Total elderly population | ≤60 | 1.00 (7.9) | 1.00 (23.7) | 1.00 (6.4) | 1.00 (21.5) |
| | 61 to 80 | 0.90 | 0.96 | 0.95 | 0.91 |
| | 81 to 365 | 1.28 | 1.07 | 1.11 | 0.96 |

[a] Base rates are given in parentheses.

[b] Relative risk is based on less than 500 person-days and is considered unstable.

## Table 5.5.8. RELATIVE RISK OF CARDIOPULMONARY SYMPTOM AGGRAVATION IN ELDERLY PANELISTS EXPOSED TO VARYING LEVELS OF TOTAL SUSPENDED PARTICULATES[a]

| Panel | TSP concentration (24 hr), $\mu g/m^3$ | $T_{min}$ = 20 to 40°F | | $T_{min}$ > 40°F | |
|---|---|---|---|---|---|
| | | Low | Pooled Intermediate | Low | Pooled Intermediate |
| Well | ≤60 | 1.00 (8.1) | 1.00 (18.3) | 1.00 (6.1) | 1.00 (14.3) |
| | 61 to 75 | 1.23[b] | 0.98 | 1.15[b] | 0.94 |
| | 76 to 260 | 0.83[b] | 0.99 | 0.66[b] | 1.10 |
| Heart | ≤60 | 1.00 (5.6) | 1.00 (39.0) | 1.00 (5.0) | 1.00 (33.6) |
| | 61 to 75 | 0.45[b] | 0.95 | 0.60[b] | 1.04 |
| | 76 to 260 | 0.36[b] | 0.99 | 1.20[b] | 0.99 |
| Lung | ≤60 | 1.00 (12.2) | 1.00 (29.1) | 1.00 (16.5) | 1.00 (22.6) |
| | 61 to 75 | 0.94[b] | 1.04 | 1.03[b] | 1.18[b] |
| | 76 to 260 | 0.96[b] | 1.06 | 0.85[b] | 1.01 |
| Heart and lung | ≤60 | 1.00 (21.8) | 1.00 (51.2) | 1.00 (16.0) | 1.00 (47.5) |
| | 61 to 75 | 1.58[b] | 1.02 | 0.42[b] | 1.04 |
| | 76 to 260 | 1.65[b] | 1.01 | 1.25 | 0.96 |
| Total elderly population | ≤60 | 1.00 (8.1) | 1.00 (24.2) | 1.00 (6.9) | 1.00 (20.9) |
| | 61 to 75 | 1.04 | 0.94 | 1.01 | 0.98 |
| | 76 to 260 | 0.75 | 0.99 | 0.80 | 1.07 |

[a] Base rates are given in parentheses.

[b] Relative risk is based on less than 500 person-days and is considered unstable.

of increase in "heart" and "well" panels on the warmer days. It was difficult, however, to relate unequivocal adverse health effects to the levels of suspended particulate observed during this study.

Suspended particulate sulfate exposures were significantly correlated with adverse health effects (Table 5.5.9). In the Intermediate communities, elevated sulfate levels were consistently related to increments in symptom rates in all panels except the "heart" panel on colder days. In general, the relative risks for the Low exposure community reflected the same trend, even though they were themselves somewhat erratic. There was good evidence that a threshold effect existed somewhere between 6 and 10 μg/m³, with the sickest panel being the most susceptible. The best estimates for the total elderly population predicted that annual average suspended sulfate levels of 10 to 12 μg/m³ would be accompanied by a 6 percent morbidity excess on colder days and a 32 percent morbidity excess on warmer days.

In summary, temperature-specific relative risk analyses confirmed that the sickest ("heart and lung") panelists were more likely to report exacerbations of their symptoms and that suspended sulfates were the strongest contributory pollutant determinant. The relationships for the three pollutants considered are illustrated in Figure 5.5.7 for "heart and lung" panelists.

**Table 5.5.9. RELATIVE RISK OF CARDIOPULMONARY SYMPTOM AGGRAVATION IN ELDERLY PANELISTS EXPOSED TO VARYING LEVELS OF SUSPENDED SULFATE[a]**

| Panel | SS concentration (24 hr), μg/m³ | $T_{min}$ = 20 to 40°F | | $T_{min}$ > 40°F | |
|---|---|---|---|---|---|
| | | Low | Pooled Intermediate | Low | Pooled Intermediate |
| Well | ≤6.0 | 1.00 (8.1) | 1.00 (17.2) | 1.00 (6.3) | 1.00 (10.9) |
| | 6.1 to 8 | 1.07 | 0.86 | 0.83 | 1.24 |
| | 8.1 to 10 | 0.90 | 1.08 | 0.97 | 1.25 |
| | 10.1 to 12 | 1.10 | 1.08 | 0.73[b] | 1.44 |
| | >12 | 0.99 | 1.12 | 1.08 | 1.37 |
| Heart | ≤6.0 | 1.00 (5.7) | 1.00 (41.7) | 1.00 (4.7) | 1.00 (36.0) |
| | 6.1 to 8 | 1.27 | 0.90 | 1.09[b] | 0.99 |
| | 8.1 to 10 | 0.81[b] | 0.97 | 0.98 | 1.04 |
| | 10.1 to 12 | 0.93 | 0.93 | 1.02[b] | 1.09 |
| | >12 | 0.86 | 1.00 | 1.21[b] | 1.19 |
| Lung | ≤6.0 | 1.00 (11.2) | 1.00[b] (27.3) | 1.00 (12.6) | 1.00 (23.2) |
| | 6.1 to 8 | 1.12 | 0.92 | 0.97[b] | 1.07 |
| | 8.1 to 10 | 1.07[b] | 1.06 | 0.79[b] | 1.00 |
| | 10.1 to 12 | 1.24 | 1.20[b] | 1.41[b] | 0.93 |
| | >12 | 1.08 | 1.10 | 1.15[b] | 1.20 |
| Heart and lung | ≤6.0 | 1.00[b] (18.7) | 1.00[b] (51.3) | 1.00[b] (12.9) | 1.00 (46.8) |
| | 6.1 to 8 | 1.00[b] | 1.08 | 1.40[b] | 1.01 |
| | 8.1 to 10 | 1.02[b] | 1.16 | 1.56[b] | 1.02 |
| | 10.1 to 12 | 1.35[b] | 1.16 | 1.16[b] | 1.17 |
| | >12 | 1.53[b] | 1.15 | 1.22[b] | 1.11 |
| Total elderly population | ≤6.0 | 1.00 (8.0) | 1.00 (24.0) | 1.00 (6.6) | 1.00 (18.0) |
| | 6.1 to 8 | 1.12 | 0.88 | 0.91 | 1.17 |
| | 8.1 to 10 | 0.90 | 1.06 | 0.97 | 1.18 |
| | 10.1 to 12 | 1.08 | 1.06 | 0.86 | 1.32 |
| | >12 | 0.98 | 1.09 | 1.12 | 1.31 |

[a] Base rates are given in parentheses.

[b] Relative risk is based on less than 500 person-days and is considered unstable.

Overall, morbidity excesses in the elderly population could be predicted when increased exposures to suspended sulfates occurred.

Excess risk models were used to convey the importance of ambient temperature alone and of suspended sulfates alone in the exacerbation of symptoms and to relate pollutant-induced risks to a more familiar effect like seasonal fluctuation in temperature (Table 5.5.10). Excess morbidity attributable to normal seasonal temperature fluctuations usually somewhat exceeded that attributable to suspended sulfates.

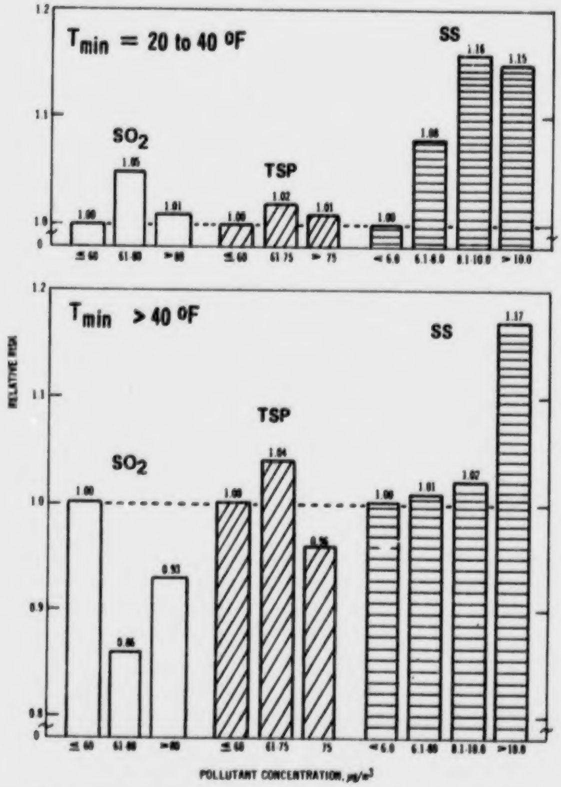

Figure 5.5.7. Relative risk of symptom aggravation versus sulfur dioxide, total suspended particulate, and suspended sulfate concentrations: two minimum temperature ranges, "heart and lung" panel.

## Threshold and Linear Dose-response Functions

In setting air quality standards, the existence of a threshold concentration for a health effect is implicitly assumed. It is important, therefore, to ascertain if the dose-response relationship can be reasonably estimated by a function indicating no response until some nonzero threshold concentration is exceeded. Most statistical procedures assume a strictly increasing functional relationship between two variables, and therefore that a significant association exists at all pollutant levels, no matter how low.

As a simple alternative, we hypothesized, for a threshold function, a segmented line having a zero slope below exposure level x and a positive slope at levels above x. The point x is estimated by the least squares method. This function has been designated a "hockey stick" function.[19] This least squares technique had been earlier applied to other more general problems.[20] The purpose is to estimate the threshold, not the entire dose-response function.

Temperature-specific threshold functions were computed using the experience of the pooled populations from the Intermediate communities (Table 5.5.11). Illustrative plots of threshold functions for the "heart and lung" panel for each of three pollutants are presented in Figure 5.5.8. Only one threshold for sulfur dioxide could be estimated, 181 $\mu g/m^3$ for the sickest panel on the colder days ($T_{min}$ = 20 to 40 °F). For total suspended particulates, threshold estimates were fitted for symptom induction in the "well" panel (68 $\mu g/m^3$) on warmer days ($T_{min} > 40$ °F) and in the "heart and lung" panel (47 $\mu g/m^3$) on cooler days ($T_{min}$ = 20 to 40 °F). For suspended sulfates, one or more threshold estimates could be made for every panel. The estimate for the "heart and lung" panel on the colder days was 9.2 $\mu g/m^3$. On warmer days, the elderly in general and the "lung" panel in particular tolerated higher sulfate thresholds (10.9 $\mu g/m^3$) than on cold days when the threshold was zero. For the other two panels, computed threshold estimates ranged from 0 to 9.8. Confidence intervals overlapped for most of the point estimates of suspended sulfate effects. Overall, suspended sulfate levels of 2 to 6 $\mu g/m^3$ could be assumed to represent a reasonable threshold on colder days while 9 to 10 $\mu g/m^3$ would be a reasonable overall effects threshold estimate for warmer days.

### Table 5.5.10. EXCESS CARDIOPULMONARY SYMPTOMS IN ELDERLY PANELISTS ATTRIBUTABLE TO TEMPERATURE AND TO SUSPENDED SULFATE EXPOSURES

| Panel | Community | Percent excess attributable to | | |
|---|---|---|---|---|
| | | Colder temperatures[a] | Suspended sulfates[b] | |
| | | | $T_{min}$ = 20 to 40°F | $T_{min}$ > 40°F |
| Well | Low | 29 | 10 | -27[c] |
| | Intermediate | 58 | 8 | 44 |
| Heart | Low | 21 | -7 | 2[c] |
| | Intermediate | 16 | -7 | 9 |
| Lung | Low | -12 | 24 | 41[c] |
| | Intermediate | 29 | 20[c] | -7 |
| Heart and lung | Low | 36 | 35[c] | 16[c] |
| | Intermediate | 16 | 16 | 17 |
| Total elderly population | Low | 17 | 8 | -14 |
| | Intermediate | 16 | 6 | -32 |

[a] Percent excess on cool days ($T_{min}$ = 20 to 40°F) with low pollution levels as compared with warmer days ($T_{min}$ > 40°F) with low pollution levels.

[b] Percent excess on days with suspended sulfate concentrations of 10.1 to 12 $\mu g/m^3$ as compared with days of 6.0 $\mu g/m^3$ or less.

[c] Relative risk is based on less than 500 person-days and is considered unstable.

### Table 5.5.11. TEMPERATURE-SPECIFIC THRESHOLD ESTIMATES FOR THE EFFECT OF SELECTED POLLUTANTS ON ELDERLY PANELISTS REPORTING CARDIOPULMONARY SYMPTOMS

| Pollutant and panel | Minimum daily temperature ($T_{min}$), °F | Intercept (aggravation rate per person) | Estimated effects threshold, $\mu g/m^3$ | Slope | Percent of days threshold exceeded in N.Y. in 1971 | Estimated percent excess risk at presently allowable once-a-year levels[a] |
|---|---|---|---|---|---|---|
| Sulfur dioxide | | | | | | |
| Heart and lung | 20 to 40 | 0.53 | 181 | 0.001352 | <5 | 47 |
| Total suspended particulates | | | | | | |
| Well | >40 | 0.14 | 68 | 0.000456 | 50 to 60 | 63 |
| Heart and lung | 20 to 40 | 0.51 | 47 | 0.000880 | 70 to 80 | 37 |
| Suspended sulfates | | | | | | |
| Well | 20 to 40 | 0.17 | 0 | 0.001132 | 100 | 33 |
| | >40 | 0.13 | 1.9 | 0.001769 | 100 | 65 |
| Heart | >40 | 0.37 | 9.8 | 0.007817 | 70 | 85 |
| Lung | 20 to 40 | 0.25 | 0 | 0.003479 | 100 | 70 |
| | >40 | 0.22 | 10.9 | 0.007557 | 50 | 134 |
| Heart and lung | 20 to 40 | 0.51 | 9.2 | 0.003277 | 70 | 26 |

[a] Suspended sulfate level of 50 $ug/m^3$, which occurs infrequently in New York, was chosen arbitrarily since no standard exists.

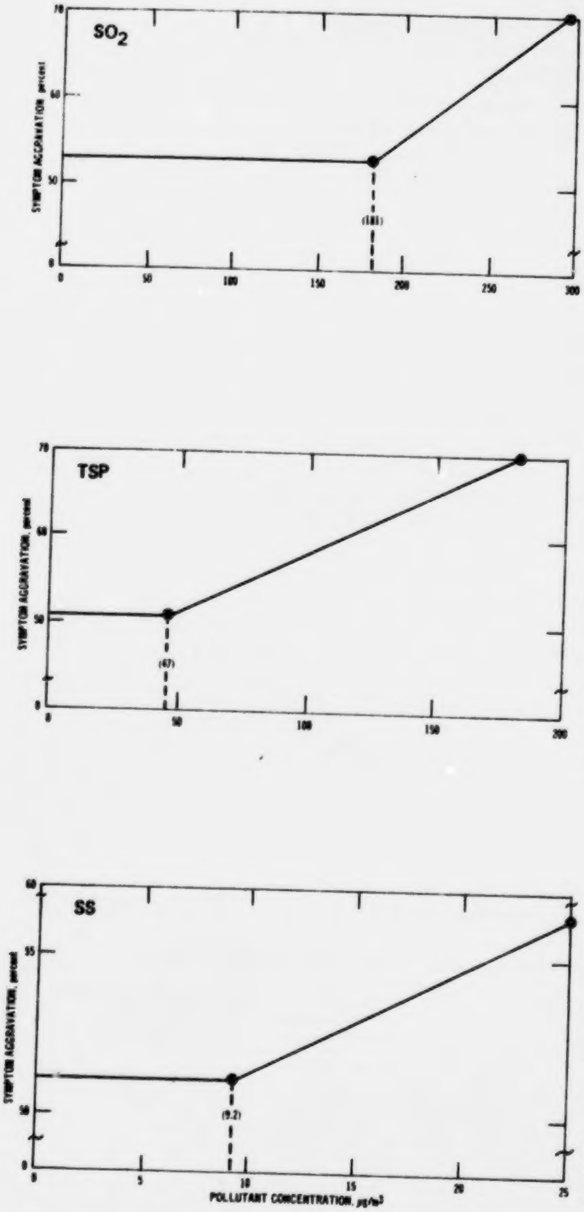

Figure 5.5.8. Temperature-specific threshold estimates for symptom aggravation by sulfur dioxide, total suspended particulates, and suspended sulfates: minimum temperature, 20 to 40 °F; "heart and lung" panel.

## Implications of Threshold Estimates

Threshold estimates and their health and air quality implications are summarized in Table 5.5.11. If one accepts 181 $\mu g/m^3$ as a sulfur dioxide threshold on colder days, New York study areas now exceed this level on less than 5 percent of the days during each year. However, if sulfur dioxide levels climbed as high as the currently permissible once yearly 24-hour level (365 $\mu g/m^3$), a substantial morbidity excess, 47 percent over the base, might be expected. For total suspended particulates, the relevant threshold levels are exceeded on 50 to 80 percent of the days during each year and morbidity excesses of 37 to 63 percent might be expected among elderly groups. For suspended sulfates, the low-temperature thresholds are exceeded every day; the thresholds for warmer days are exceeded on 50 to 70 percent of all days. Morbidity excesses at peak 24-hour exposures currently monitored ($\sim$50 $\mu g/m^3$) would range from 26 to 134 percent. These findings suggest that short-term exposures involving sulfur dioxide, total suspended particulates, and, more importantly, suspended sulfates will adversely affect the health of our senior citizens even after the current urban air quality goals for sulfur dioxide and particulates are achieved.

## DISCUSSION

All three goals of the study were at least partially attained. Three air pollutants were found to be associated with aggravation of chronic cardiopulmonary disease symptoms in panels of elderly patients. These pollutants were sulfur dioxide, total suspended particulates, and, most strongly, suspended particulate sulfates. Conservative estimates utilizing patterns of significance in multiple regression analyses suggested that most, if not all, of the adverse effects could be attributed to suspended sulfates since no significant residual effects were noted after the effects of ambient temperature and suspended sulfates were removed. Temperature-specific relative risk estimates and threshold calculations are more likely to attribute a significant role to any single pollutant than the more conservative multiple regression analyses. Such was the case, with all pollutants causing aggravation of symptoms in the most vulnerable, sickest panelists. Again, temperature-specific analyses assigned larger and more consistent effects and lower thresholds to suspended sulfates than to the other two pollutants.

Unexpectedly high levels of suspended sulfates in the Low exposure community provided a double-blind study of the effects of this pollutant. Despite the relatively small panel sizes and fewer polluted days, elderly panelists from this community generally reported higher symptom rates on days when sulfate levels exceeded 10.0 $\mu g/m^3$. Furthermore, the total elderly population of panelists from the Low exposure community had estimated higher symptom rates on days when sulfur dioxide exceeded 80 $\mu g/m^3$ than on less polluted days. The deleterious effects of suspended sulfates in the Low exposure community were significant in both the multiple regression and temperature-specific analyses.

The effects of short-term peak exposures in today's urban air should be placed in a broader perspective. Air quality has improved, suggesting that peak exposures and deleterious health effects on patients with chronic heart and lung disorders might have been much greater in the past. Moreover the pollutant effects now observed in one of our largest metropolitan areas are generally of the same magnitude as deleterious effects of normally accepted seasonal and short-term changes in ambient temperature. The estimated threshold effects for suspended particulates and suspended sulfates are close to the observed background levels monitored in remote locations. Analyses reported here, however, imply that major, potentially life-threatening health impairments in our large elderly population can be related to commonly monitored levels of urban pollutants. Furthermore, modern heating and air conditioning, to some extent, are ameliorating the very large mortality and morbidity effects of wide swings in ambient temperature. Therefore, if urban air pollution is not well controlled and the use of central heating and air conditioning continues to increase, pollutant effects could easily surpass those now attributed to ambient temperature.

Several problems that arose in the present study must be considered, including intercommunity differences, difficulties in panelist recruitment, panelist participation, and the role of indoor air pollutants. Panelists from the Intermediate communities differed intrinsically from panelists in the Low exposure community; pollutant effects could be identified only by time series analyses within each exposure community. Panelists from the Intermediate communities were also somewhat less faithful participants. The utilization of elderly panelists for the study of day-to-day changes in disease symptoms involved many difficulties. Initial recruitment posed the problem of finding elderly people with heart and/or lung disease who would agree to participate in the study. Recruitment was very time-consuming as only about 5 percent of those interviewed both met our criteria and agreed to participate. Once recruited, motivation became a major problem. Since the potential benefits of pollution control may not be realized by these people and we provided no special incentive for them to assure their participation, it proved difficult to maintain their interest. Many of the most elderly found questionnaires difficult to understand; thus, frequent explanations were necessary to assure that the panelists filled in their diaries daily rather than at the end of the week. Many panelists found the study too bothersome in requiring maintenance of a daily diary for 32 weeks, and there was a marked decrease in active participants over the course of the study. The questions asked on a daily basis also contributed to the difficulty of this study. Recruitment of panelists with lung disease was contingent solely upon a history of cough, phlegm, or both for more than 3 months out of a year. Thus, all "lung" and "heart and lung" panelists could potentially vary in this symptom, and therefore each diary response was valid for evaluation of daily symptom change. However, only 38 percent of all "lung" panelists had a history of shortness of breath, so that daily variation in this symptom was based upon a limited subsample of this panel. Similarly, the background history varied in the number of panelists who could be expected to exhibit day-to-day changes in other diary symptoms. Despite these frustrating difficulties, the use of elderly panelists proved feasible and provided health intelligence needed for air quality criteria and indirectly for evaluation of National Primary Ambient Air Quality Standards.

Indoor air quality could not be evaluated in this study. However, suspended sulfates, which proved to be the pollutant that manifests the strongest relationships to cardiopulmonary symptoms, are primarily fine particulates that should penetrate indoors.[24]

After careful consideration of all the evidence, we concluded that there was preponderant evidence that fluctuations in suspended sulfate levels can be linked to significant worsening in the symptoms of patients with chronic heart and lung disease. Suspended sulfate levels commonly found in the major cities of our country ( ~10 $\mu g/m^3$) have a deleterious impact even when 24-hour sulfur dioxide and suspended particulate levels are well within the limits set by National Primary Air Quality Standards. We also believe it is possible, but not likely, that the relatively low 24-hour levels of sulfur dioxide (180 $\mu g/m^3$) and total suspended particulates (50 to 70 $\mu g/m^3$) may also have an adverse effect on the symptom status of the sickest panelists. Such levels are well below the currently acceptable upper limits for 24-hour peak exposures involving these pollutants. We could not be certain that each of these pollutants has an independent effect, but believe it prudent to assume that any of the aforementioned pollutants may result in adverse health effects in elderly populations. Repeated studies should be conducted to strengthen confidence in the threshold values presented in this report.

## SUMMARY

Over 500 elderly panelists who were well or who reported cardiopulmonary symptoms participated in a diary study linking aggravation or induction of symptoms to fluctuations in ambient air pollutants and minimum daily temperature. Panels were previously diagnosed by a physician as being well (without cardiopulmonary symptoms), having heart disease, having lung disease, or having combined heart and lung disease. Both temperature and pollutants were associated with changes in symptom status. Decreasing temperatures induced or aggravated symptoms in all but the heart disease panelists, who reported worsening of symptoms with elevations in ambient temperature. Elevated levels of sulfur dioxide, total suspended particulates, and especially suspended sulfates were linked to symptom exacerbations. However, within temperature-specific ranges, suspended sulfates exhibited the strongest and most consistent association with symptom aggravation. The best estimate of threshold levels was $\leq 10$ $\mu g/m^3$ for suspended sulfates. The implications of these thresholds in light of current air pollution levels and the relative importance of seasonal swings in temperatures were discussed, and the problem was judged to be of considerable public health significance.

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### Table 5.5.A.1. BACKGROUND INTERVIEW INFORMATION

| Panel and characteristic | Percent of enrolled panelists | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| Well | | | |
| Over 70 years old | 31 | 53 | 51 |
| Smokers | 48 | 71 | 68 |
| Males | 38 | 37 | 40 |
| Education-high school or less | 92 | 97 | 97 |
| Shortness of breath | 7 | 16 | 15 |
| Leg swelling | 15 | 25 | 16 |
| Medication, heart or water | 7 | 5 | 4 |
| Heart | | | |
| Over 70 years old | 25 | 38 | 35 |
| Smokers | 50 | 51 | 66 |
| Males | 42 | 42 | 36 |
| Education-high school or less | 88 | 94 | 97 |
| Shortness of breath | 34 | 57 | 48 |
| Leg swelling | 27 | 52 | 31 |
| Medication, heart or water | 34 | 62 | 56 |
| Angina | 27 | 21 | 21 |
| Chest pain with exertion or upset relieved by rest | 36 | 74 | 71 |
| Heart attack | 43 | 45 | 36 |
| Large heart | 34 | 21 | 20 |
| Lung | | | |
| Over 70 years old | 27 | 48 | 59 |
| Smokers | 31 | 55 | 47 |
| Males | 65 | 41 | 47 |
| Education-high school or less | 85 | 97 | 97 |
| Shortness of breath | 17 | 41 | 53 |
| Leg swelling | 14 | 28 | 24 |
| Cough and phlegm > 3 months | 35 | 13 | 15 |
| Cough and phlegm > 3 months and shortness of breath | 3 | 22 | 27 |
| Heart and lung | | | |
| Over 70 years old | 31 | 46 | 55 |
| Smokers | 56 | 56 | 49 |
| Males | 63 | 42 | 47 |
| Education-high school or less | 88 | 98 | 98 |
| Shortness of breath | 63 | 83 | 71 |
| Leg swelling | 38 | 63 | 54 |
| Medication, heart or water | 69 | 63 | 60 |
| Cough and phlegm > 3 months | 19 | 6 | 15 |
| Cough and phlegm > 3 months and shortness of breath | 31 | 46 | 29 |
| Angina | 25 | 13 | 13 |
| Chest pain with exertion or upset relieved by rest | 38 | 27 | 31 |
| Heart attack | 38 | 27 | 34 |
| Large heart | 38 | 21 | 21 |

## Table 5.5.A.2. CHANGE IN PANEL MEMBERSHIP WITH TIME

| Panel | Week of study | Number of active panel members<br>Low | Intermediate I | Intermediate II |
|---|---|---|---|---|
| Well | 1 | 103 | 82 | 144 |
|  | 30 | 74 | 38[a] | 57[a] |
| Heart | 1 | 41 | 90 | 88 |
|  | 30 | 33 | 33[a] | 32[a] |
| Lung | 1 | 32 | 33 | 41 |
|  | 30 | 23 | 13[a] | 22 |
| Heart and lung | 1 | 16 | 57 | 70 |
|  | 30 | 12 | 33 | 33[a] |
| All panels | 1 | 192 | 262 | 343 |
|  | 30 | 142 | 117[a] | 144[a] |

[a] Greater than 50 percent reduction of panel size over the course of the 30 weeks.

## Table 5.5.A.3. CORRELATION COEFFICIENTS FOR SYMPTOMS, POLLUTANTS, AND TEMPERATURE[a]

| Symptom and panel | Community | TSP | SN | SS | $SO_2$ | Max. temp | Min. temp |
|---|---|---|---|---|---|---|---|
| Shortness of breath |  |  |  |  |  |  |  |
| Lung | Low | -0.032 | 0.096 | -0.043 | -0.085 | -0.153 | -0.105 |
|  | Int. I | 0.035 | -0.130 | 0.362 | 0.175 | -0.456 | -0.383 |
|  | Int. II | 0.008 | 0.072 | -0.007 | -0.305 | 0.086 | 0.164 |
| Heart | Low | -0.088 | -0.109 | -0.074 | 0.034 | 0.122 | 0.074 |
|  | Int. I | -0.055 | 0.072 | 0.130 | -0.183 | 0.109 | 0.128 |
|  | Int. II | 0.140 | 0.098 | 0.328 | 0.073 | 0.018 | 0.036 |
| Heart and lung | Low | 0.091 | 0.098 | 0.284 | 0.036 | -0.143 | -0.132 |
|  | Int. I | 0.050 | -0.027 | 0.322 | 0.049 | -0.249 | -0.143 |
|  | Int. II | 0.013 | 0.054 | 0.115 | 0.031 | -0.075 | -0.006 |
| Cough and phlegm |  |  |  |  |  |  |  |
| Well | Low | 0.036 | -0.009 | 0.053 | 0.125 | -0.237 | -0.229 |
|  | Int. I | -0.044 | -0.139 | 0.233 | 0.031 | -0.443 | -0.433 |
|  | Int. II | 0.068 | -0.019 | 0.174 | 0.202 | -0.406 | -0.388 |
| Lung | Low | -0.039 | 0.067 | -0.008 | -0.042 | -0.168 | -0.125 |
|  | Int. I | -0.002 | -0.118 | 0.277 | 0.110 | -0.486 | -0.444 |
|  | Int. II | 0.014 | 0.155 | 0.034 | -0.288 | 0.207 | 0.249 |

### Table 5.5.A.3 (continued). CORRELATION COEFFICIENTS FOR SYMPTOMS, POLLUTANTS, AND TEMPERATURE[a]

| Symptom and panel | Community | TSP | SN | SS | $SO_2$ | Max. temp | Min. temp |
|---|---|---|---|---|---|---|---|
| Heart and lung | Low | 0.175 | 0.081 | 0.252 | 0.171 | -0.128 | -0.132 |
| | Int. I | -0.067 | -0.113 | 0.119 | -0.234 | -0.124 | -0.076 |
| | Int. II | 0.188 | 0.072 | 0.270 | 0.122 | -0.293 | -0.256 |
| Angina or chest pain | | | | | | | |
| Well | Low | 0.040 | 0.106 | -0.023 | -0.054 | -0.038 | -0.107 |
| | Int. I | -0.012 | 0.044 | 0.145 | -0.181 | -0.008 | -0.050 |
| | Int. II | -0.016 | -0.084 | 0.078 | 0.150 | -0.197 | -0.106 |
| Heart | Low | -0.046 | -0.090 | -0.038 | -0.169 | 0.198 | 0.206 |
| | Int. I | 0.044 | 0.056 | 0.136 | 0.119 | 0.065 | 0.116 |
| | Int. II | -0.085 | -0.109 | -0.001 | 0.127 | 0.130 | 0.145 |
| Heart and lung | Low | 0.067 | 0.048 | 0.166 | 0.143 | -0.224 | -0.244 |
| | Int. I | -0.032 | -0.077 | 0.205 | -0.004 | -0.079 | -0.033 |
| | Int. II | 0.110 | 0.030 | 0.200 | 0.215 | -0.184 | -0.120 |
| Wheezing | | | | | | | |
| Well | Low | 0.066 | 0.077 | 0.230 | 0.048 | -0.125 | -0.109 |
| | Int. I | -0.072 | -0.008 | 0.165 | 0.035 | -0.248 | -0.150 |
| | Int. II | -0.013 | -0.017 | -0.040 | 0.088 | -0.205 | -0.135 |
| Lung | Low | -0.116 | -0.045 | -0.092 | 0.136 | -0.094 | -0.089 |
| | Int. I | 0.113 | -0.162 | 0.295 | 0.247 | -0.593 | -0.580 |
| | Int. II | 0.130 | 0.176 | -0.050 | -0.163 | 0.001 | -0.860 |
| Leg swelling | | | | | | | |
| Heart | Low | -0.096 | 0.038 | -0.064 | 0.004 | 0.089 | 0.125 |
| | Int. I | -0.038 | 0.069 | -0.026 | 0.024 | 0.296 | 0.275 |
| | Int. II | 0.027 | 0.071 | -0.080 | -0.257 | 0.105 | 0.121 |
| Heart and lung | Low | -0.018 | 0.046 | 0.158 | -0.118 | 0.088 | 0.159 |
| | Int. I | -0.137 | -0.004 | -0.040 | -0.081 | -0.045 | -0.021 |
| | Int. II | 0.184 | 0.097 | 0.020 | -0.081 | 0.001 | -0.022 |

[a] Underlined are significant at the 5 percent level.

# 5.6 VENTILATORY FUNCTION IN SCHOOL CHILDREN: 1970-1971 NEW YORK STUDIES

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## INTRODUCTION

Previous studies in Japan, Great Britain, Italy, and North America have shown decreased lung function in children living in areas of high air pollution.[1-6] Based on these reports, lung function in school children, indexed by 0.75-second timed forced expiratory volume ($FEV_{0.75}$), was chosen as one of the health indicators in the Environmental Protection Agency's Community Health and Environmental Surveillance System (CHESS). Highly significant decreases in ventilatory function are seen with aging, chronic obstructive lung disease, and self-pollution by cigarette smoking.[7] Thus, significant decrements in ventilatory function early in life may reasonably be viewed as a risk factor for later respiratory disorders. Furthermore, cessation of self-pollution with cigarette smoke is accompanied by an amelioration of chronic respiratory disease symptoms.[8] Therefore, stringent control of ambient air pollution might be associated, over time, with improvements in defective ventilatory function and with decreased risk of chronic respiratory disease that can be attributed to earlier elevated air pollution exposures. This study was designed to test three hypotheses: (1) early childhood exposure to ambient air pollution reduces ventilatory function in later childhood, (2) improvements in air quality tend to reverse deficits in ventilatory function, and (3) monthly variations in ambient air pollution exposures are paralleled by variations in ventilatory function.

## METHODS

Three New York neighborhoods were selected to represent an air pollution gradient among communities reasonably similar in general socioeconomic characteristics. After considering long-term air pollution exposure trends, Riverhead, Long Island, was chosen as a Low exposure community, the Howard Beach section of Queens as an Intermediate exposure community, and the Westchester section of the Bronx as a High exposure community. As a result of recent improvements in air quality, the Queens and Bronx neighborhoods were found to have similar current pollution levels, often below the National Primary Air Quality Standards. Hence, the latter two communities were redesignated Intermediate I and Intermediate II, respectively. During the study months, air quality averages for both suspended particulates and sulfur dioxide were between the National Primary and National Secondary Air Quality Standards. However, during the previous 15 years, the Intermediate exposure communities had experienced annual average pollution levels that were 2 to 5 times those recorded during the present study.[9]

Within each community, children in three elementary schools, including kindergarten through sixth grades (ages 5 to 13 years) and located within 1.5 miles of a single air monitoring station, were enrolled in the study. Few children were bused into any of the participating schools. Each air monitoring station sampled air from the immediate home neighborhood of the children. Four rounds of pulmonary function testing were conducted in the schools in the late fall (November-December 1970), early winter (January 1971), late winter (February-March 1971), and spring (April 1971). The National Cylinder Gas (NCG) Pulmonary Function Indicator was used to measure 0.75-second forced expiratory volume ($FEV_{0.75}$). This instrument is small, light, easily portable, and capable of giving immediate digital read-outs of $FEV_{0.75}$, $FEV_{1.0}$, and vital capacity. The instrument measures air flow by means of a change in temperature across a heat-sensitive platinum element within a transducer. Air flow is electronically integrated over time to produce a volume measurement. The pulmonary function indicators were calibrated against a Stead-Wells volume spirometer. They were tested for reproducibility by obtaining six or seven successive $FEV_{0.75}$ measurements for each indicator with trained subjects and comparing these measurements with those obtained with a Stead-Wells spirometer connected in series. Percent differences between the pulmonary function indicator and the spirometer for 25 trials ranged from -7.0 to +6.6 and averaged -0.7 (Table 5.6.1).

Four teams, trained to obtain FEV measurements from school children, collected data simultaneously; each team was rotated across all areas. Each pulmonary function indicator was used a similar number of times in each community. Age, sex, and race data were obtained from school records and verified at the time of testing. On the day of each test, standing height was measured and children were queried about the presence of cold, cough, or sore throat. Each child was tested until two readings agreed within 10 percent of the child's best performance. The maximum of the two best results was used in the analysis.

Measurements of total suspended particulates, suspended nitrates, and suspended sulfates were obtained daily using a high-volume sampler. Gas bubblers were used to monitor nitrogen dioxide by the Jacobs-Hochheiser method and sulfur dioxide by the West-Gaeke method. Soiling index was recorded at 2-hour intervals on tape samplers.[9]

Two aspects of the location of the monitoring sites must be considered in relating these air quality data to the exposure of these communities. First, the air monitoring station in the Intermediate II community was situated on top of a three-story court house in the center of a busy commercial area, while those in the Intermediate I and Low communities were located on two-story buildings surrounded by residential dwellings. Thus, at the Intermediate II station, proximity to heavy traffic would tend to increase measured pollutant levels, while greater height above ground would tend to decrease these values. The stations in the Intermediate I and Low communities probably more accurately reflected the ambient exposure experienced by nearby residents. Secondly, the Intermediate I community lies about 1 mile west of the John F. Kennedy International Airport; odors and noise from the airport have been the object of many complaints by the residents of that community. Because of budget limitations, hydrocarbons, one of the important pollutant classes

**Table 5.6.1. CALIBRATION OF PULMONARY FUNCTION INDICATOR AGAINST STEAD-WELLS SPIROMETER**

| Pulmonary function indicator | Trial | $FEV_{0.75}$, liters PFI | $FEV_{0.75}$, liters Stead-Wells | Percent difference | Average percent difference |
|---|---|---|---|---|---|
| 1 | 1 | 3.01 | 2.99 | +0.7 | |
| | 2 | 1.40 | 1.44 | -2.8 | |
| | 3 | 2.82 | 2.85 | -1.1 | |
| | 4 | 2.86 | 2.85 | +0.4 | |
| | 5 | 1.74 | 1.82 | -4.4 | |
| | 6 | 2.68 | 2.65 | +1.1 | -1.0 |
| 2 | 1 | 3.33 | 3.58 | -7.0 | |
| | 2 | 3.41 | 3.53 | -3.4 | |
| | 3 | 3.38 | 3.54 | -4.5 | |
| | 4 | 3.50 | 3.46 | +1.2 | |
| | 5 | 3.52 | 3.63 | -3.0 | |
| | 6 | 3.64 | 3.67 | -0.8 | -2.9 |
| 3 | 1 | 3.00 | 3.08 | -2.6 | |
| | 2 | 3.00 | 3.04 | -1.3 | |
| | 3 | 3.03 | 2.95 | +2.7 | |
| | 4 | 3.27 | 3.21 | +1.9 | |
| | 5 | 3.24 | 3.11 | +4.2 | |
| | 6 | 3.16 | 3.10 | +1.9 | +1.1 |
| 4 | 1 | 3.02 | 3.14 | -3.8 | |
| | 2 | 2.83 | 2.93 | -3.4 | |
| | 3 | 3.06 | 3.08 | -0.6 | |
| | 4 | 2.81 | 2.84 | -1.1 | |
| | 5 | 2.51 | 2.44 | +2.9 | |
| | 6 | 2.09 | 1.96 | +6.6 | |
| | 7 | 2.02 | 2.02 | +0.0 | +0.1 |
| Overall average | | | | | -0.7 |

emitted by aircraft engines, were not monitored at any of the CHESS stations during the 1970-1971 study period. Therefore, the influence of aircraft emissions on the exposure of residents could not be determined from the pollutant data available.

## RESULTS

### Population Characteristics

An analysis of socioeconomic characteristics of the families of children participating in the study, documented through personal interviews of a sample of families, showed only slight differences between study areas:

1. Age - same distribution in all areas.
2. Income - slightly higher in the Intermediate I community and lower in the Intermediate II community when compared with the Low exposure community.
3. Education - slightly higher in the Intermediate I community and lower in the Intermediate II community when compared with the Low exposure community.
4. Duration of residence - slightly shorter in the Intermediate I community, but mobility occurred principally within a single area.
5. Size of family - somewhat larger in the Low exposure community.
6. Smoking by parents - no appreciable difference among communities.

### Pollutant Exposures

Estimates of air pollution exposure during the lifetime of the study population, that is, the 12 years preceding the study, were constructed from monitoring records of the New York City Department of Air Resources (Table 5.6.2). These estimates indicated that New York City children aged 9 to 13 years at the time of study had been exposed to high levels of sulfur dioxide (about 364 to 435 $\mu g/m^3$) for the first 5 to 10 years of life. Children 5 to 8 years old were exposed to these higher levels for a shorter period of time, 5 years or less. Annual suspended sulfate levels were also high during the decade prior to the study (estimated at 9 to 25 $\mu g/m^3$). Both age groups were exposed to rapidly decreasing pollution levels for 3 years prior to the study, with sulfur dioxide levels being reduced by about three-fifths and suspended particulates by one-third. The decrease for suspended

Table 5.6.2. ESTIMATED AIR POLLUTION EXPOSURES OF STUDY COMMUNITIES DURING YEARS PRIOR TO VENTILATORY FUNCTION TESTING

| Pollutant | Community | Estimated annual average exposure[a] | | | | |
|---|---|---|---|---|---|---|
| | | 1958-1962 | 1963-1967 | 1968 | 1969 | 1970 |
| Sulfur dioxide, $\mu g/m^3$ | Low (Riverhead) | NA | NA | NA | NA | NA |
| | Intermediate I (Queens) | 424 | 425 | 233 | 131 | 157 |
| | Intermediate II (Bronx) | 364 | 435 | 320 | 236 | 184 |
| Total suspended particulates, $\mu g/m^3$ | Low | ∿35 | ∿35 | 52 | 31 | 39 |
| | Intermediate I | 200 | 158 | 92 | 75 | 85 |
| | Intermediate II | 172 | 164 | 117 | 97 | 111 |
| Suspended sulfates, $\mu g/m^3$ | Low | NA | NA | NA | NA | NA |
| | Intermediate I | 25 | 16 | 9 | 11 | 5 |
| | Intermediate II | 22 | 16 | 12 | 10 | 22 |
| Dustfall, $g/m^2/mo$ | Low | NA | NA | NA | 3 | 2 |
| | Intermediate I | 23 | 14 | 8 | 6 | 6 |
| | Intermediate II | 20 | 14 | 11 | 10 | 8 |

[a] NA–not available.

sulfates, which averaged an estimated 5 to 22 $\mu g/m^3$, was not as consistent.

During the period of ventilatory testing, the Intermediate II area was exposed to marginally higher levels of suspended particulates and sulfur dioxide than was the Intermediate I area (Table 5.6.3). As expected, both of these communities were found to have pollution levels significantly higher than the Low exposure area, where pollutant levels were substantially below the National Primary and Secondary Air Quality Standards for sulfur dioxide and suspended particulates. Concentrations in the Low exposure area tended to be about one-half those measured in the Intermediate exposure areas. The data suggest that the Intermediate exposure areas may be considered in the same pollutant category and that the Low exposure community represented a much cleaner area. Concentrations of sulfur dioxide and particulates were highest in all areas during January.

**Table 5.6.3. ARITHMETIC AVERAGE POLLUTANT CONCENTRATIONS FOR STUDY AREAS, NOVEMBER 1970 TO MAY 1971**

| Pollutant | Period | Exposure | | |
|---|---|---|---|---|
| | | Low | Intermediate I | Intermediate II |
| Total suspended particulate, $\mu g/m^3$ | Nov-Dec | 35 | 65 | 62 |
| | Jan | 43 | 69 | 83 |
| | Feb-Mar | 29 | 55 | 81 |
| | Apr-May | 30 | 59 | 72 |
| | Avg | 33 | 61 | 74 |
| Suspended nitrates, $\mu g/m^3$ | Nov-Dec | 1.6 | 1.9 | 1.5 |
| | Jan | 1.8 | 1.8 | 1.9 |
| | Feb-Mar | 1.1 | 1.6 | 2.0 |
| | Apr-May | 1.1 | 2.2 | 2.6 |
| | Avg | 1.3 | 1.9 | 2.0 |
| Suspended sulfates, $\mu g/m^3$ | Nov-Dec | 13 | 16 | 14 |
| | Jan | 15 | 17 | 18 |
| | Feb-Mar | 8 | 12 | 13 |
| | Apr-May | 7 | 9 | 9 |
| | Avg | 10 | 13 | 13 |
| Sulfur dioxide, $\mu g/m^3$ | Nov-Dec | 27 | 75 | 95 |
| | Jan | 45 | 105 | 147 |
| | Feb-Mar | 26 | 39 | 39 |
| | Apr-May | 25 | 62 | 59 |
| | Avg | 29 | 66 | 76 |
| Soiling, COHs | Nov-Dec | 0.38 | 0.70 | 1.02 |
| | Jan | 0.51 | 1.05 | 1.57 |
| | Feb-Mar | 0.36 | 0.62 | 1.01 |
| | Apr-May | 0.199 | 0.51 | 0.91 |
| | Avg | 0.34 | 0.68 | 1.06 |

On the basis of the Jacobs-Hochheiser nitrogen dioxide measurements, there was reason to conclude that exposures to nitrogen dioxide were similar in the Intermediate communities, and substantially lower in the Low exposure community. However, due to the unreliability of the Jacobs-Hochheiser method, quantitative descriptions of nitrogen dioxide exposure were not warranted.

## Ventilatory Function

Only white children present for all four testing periods were included in data analysis. Black children were excluded since their sample sizes in the Intermediate communities were too small to allow significance testing. Mean height- and age-adjusted $FEV_{0.75}$ values for each of the four test periods are presented in Figure 5.6.1 for the 2364 white children who participated in all four tests. These means were obtained from 587 children in the Low exposure area, 835 in the Intermediate I area, and 942 in the Intermediate

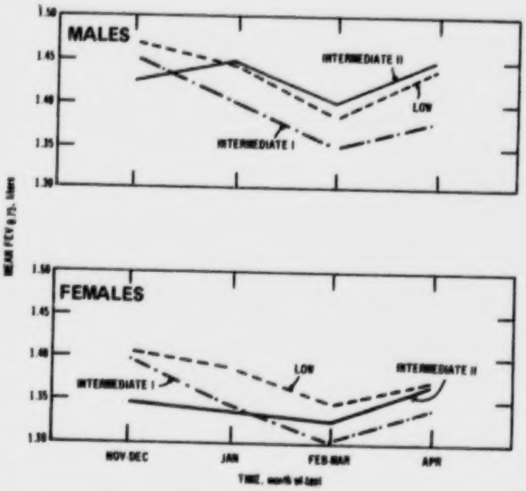

Figure 5.6.1. Height- and age-adjusted mean $FEV_{0.75}$ for males and females in each community.

II area. Month-to-month variations in $FEV_{0.75}$ values generally followed the same pattern for both sexes in the three study areas. Pulmonary function was consistently lowest in all areas in the February-March test period. The temporal pattern of $FEV_{0.75}$ results was unrelated to variations in monthly average pollutant concentrations (Table 5.6.3). However, variations in monthly means of daily minimum temperatures (Table 5.6.4) may have influenced the overall response. In general, ventilatory function was best in November, December, and April when temperatures were somewhat higher than the winter months of January through March. However, ventilatory function during January, the coldest month, was better than in February and March, which were warmer.

Except for the first period of testing, children in the Intermediate I area consistently had the poorest ventilatory performance. During the first period, lowest pulmonary function was recorded in Intermediate II area children; performance of children from the Intermediate I area was significantly higher, and performance of children from the Low exposure area was highest but relatively close to that of those from the Intermediate I area. For the remaining three test periods, males from the Intermediate II community had higher $FEV_{0.75}$ readings than males from the Low exposure area, while the opposite was true for females.

An analysis of covariance was employed to test the significance of identified important determinants of $FEV_{0.75}$, including sex, age, height, and area of residence. Significance tests were computed for each test period and for all periods combined. These analyses showed strong effects of sex, age, and height on $FEV_{0.75}$ of school children. A consistent $FEV_{0.75}$ difference between the Low exposure community and the combined Intermediate I and Intermediate II communities appeared in each of the four test periods. This difference was explained mostly by the low performance of children of both sexes living in the Intermediate I exposure area. Except for the first of the four test periods, the performance of children from the Intermediate II and Low exposure communities did not significantly differ when $FEV_{0.75}$ values were averaged across both sexes.

Since the cumulative dose of air pollution exposure of younger children (5 to 8 years old) was less than that of older children (9 to 13 years old), separate analyses were performed, using the previously described procedure, to test the hypothesis that older children from the polluted areas would demonstrate a greater decrement in ventilatory function than younger children from those areas. Sex- and age group-specific height- and age-adjusted $FEV_{0.75}$ for each exposure area and each time period were calculated (Table 5.6.5). No consistent intercommunity differences in ventilatory function were found for younger children. There was a modest but steady improvement over time in ventilatory function among young males living in the Intermediate II exposure area. A similar trend, considerably dampened, was suggested for young females from the same community.

On the other hand, when all testing periods were grouped together, older children of both sexes from each of the Intermediate communities had lower ventilatory function than children in the Low exposure community. Decrements were modest, ranging from 2 to 3.4 percent, and were significant only for males (Table 5.6.6). When each testing period was considered separately, significant decrements in ventilatory function were often found for older children residing in the Intermediate communities. On two occasions, a single sex group of older children from one of

**Table 5.6.4. MONTHLY AVERAGE DAILY MINIMUM TEMPERATURE (°F) FOR EACH STUDY AREA DURING PULMONARY FUNCTION TESTING**

| Community | Nov | Dec | Jan | Feb | Mar | Apr |
|---|---|---|---|---|---|---|
| Low | 40.4 | 29.1 | 20.4 | 26.5 | 31.7 | 36.5 |
| Intermediate I | 42.4 | 29.5 | 21.7 | 28.5 | 32.8 | 39.7 |
| Intermediate II | 44.1 | 31.2 | 23.1 | 30.9 | 34.0 | 41.6 |

**Table 5.6.5. SEX- AND AGE GROUP-SPECIFIC AGE- AND HEIGHT-ADJUSTED MEAN FORCED EXPIRATORY VOLUME ($FEV_{0.75}$) DISTRIBUTED BY TEST PERIOD AND COMMUNITY**

| Test period and community | Height-adjusted $FEV_{0.75}$, liters | | | |
|---|---|---|---|---|
| | Males | | Females | |
| | 5 to 8 yr old | 9 to 13 yr old | 5 to 8 yr old | 9 to 13 yr old |
| Nov-Dec | | | | |
| Low | 1.204 | 1.821 | 1.222 | 1.698 |
| Intermediate I | 1.171 | 1.792 | 1.082 | 1.738 |
| Intermediate II | 1.193 | 1.726 | 1.065 | 1.647 |
| Jan | | | | |
| Low | 1.178 | 1.776 | 1.087 | 1.701 |
| Intermediate I | 1.196 | 1.708 | 1.091 | 1.602 |
| Intermediate II | 1.203 | 1.793 | 1.091 | 1.690 |
| Feb-Mar | | | | |
| Low | 1.141 | 1.703 | 1.049 | 1.643 |
| Intermediate I | 1.134 | 1.649 | 1.031 | 1.604 |
| Intermediate II | 1.203 | 1.682 | 1.066 | 1.593 |
| Apr | | | | |
| Low | 1.210 | 1.747 | 1.093 | 1.689 |
| Intermediate I | 1.191 | 1.660 | 1.086 | 1.621 |
| Intermediate II | 1.243 | 1.718 | 1.106 | 1.656 |
| Average | | | | |
| Low | 1.183 | 1.762 | 1.088 | 1.683 |
| Intermediate I | 1.173 | 1.702 | 1.072 | 1.641 |
| Intermediate II | 1.211 | 1.730 | 1.082 | 1.647 |
| (Sample size) | (645) | (582) | (596) | (541) |

the Intermediate communities had ventilatory performance that was higher than in the Low exposure community. On one other occasion, the decrements for older children observed in the Intermediate communities were not significant.

As expected, height and age differences were both strong determinants of ventilatory function for younger and older children. The regression coefficients for age and height by sex and age group are given in Table 5.6.7 since they may be of value to other investigators.

## DISCUSSION

White children aged 9 to 13 years living in fairly polluted (Intermediate) communities were found to have decreased ventilatory function when compared to similar children from a relatively clean (Low) community. For males, these decrements were statistically significant; for females they were not. These decrements were replicated for both sexes over time, with only two of ten possible community comparisons yielding anomalous trends. Younger children (5 to 8 years old) from polluted communities, who were relatively less exposed than their older (9 to 13) classmates, did not exhibit any decrement in ventilatory function that was attributable to air pollution. In fact, in young males from the area with the highest recent pollution exposures, a trend towards improving ventilatory function was seen during the school year. A less marked but perceptible improvement trend was noted for females from the same community.

Table 5.6.6. ANALYSIS OF VARIANCE FOR EACH TEST PERIOD BY SEX AND BY AGE GROUP (F VALUES)

| Factor | Degrees of freedom | Nov-Dec | Jan | Feb-Mar | Apr | Average |
|---|---|---|---|---|---|---|
| Males 5 to 8 years | | | | | | |
| Age | 1 | 36.11[a] | 9.82[a] | 5.46[b] | 47.03[a] | 34.88[a] |
| Height | 1 | 200.19[a] | 213.48[a] | 256.59[a] | 180.23[a] | 339.31[a] |
| Area | 2 | 1.81 | 0.80 | 11.04[a] | 7.00[a] | 5.23[a] |
| Ethnic differences | 1 | 2.37 | 2.00 | 1.83 | 0.43 | 1.22 |
| Females 5 to 8 years | | | | | | |
| Age | 1 | 37.64[a] | 11.76[a] | 17.35[a] | 28.31[a] | 37.77[a] |
| Height | 1 | 168.36[a] | 118.56[a] | 167.47[a] | 156.56[a] | 240.83[a] |
| Area | 2 | 4.58[b] | 0.03 | 2.27 | 0.50 | 0.48 |
| Ethnic differences | 1 | 0.01 | 0.21 | 0.01 | 1.85 | 0.10 |
| Males 9 to 13 years | | | | | | |
| Age | 1 | 7.23[a] | 3.38 | 1.55 | 3.26 | 4.87[b] |
| Height | 1 | 324.73[a] | 308.02[a] | 289.17[a] | 277.91[a] | 412.26[a] |
| Area | 2 | 5.89[a] | 14.73[a] | 4.62[a] | 5.24[a] | 3.71[b] |
| Ethnic differences | 1 | 0.04 | 0.78 | 0.10 | 0.43 | 0.24 |
| Females 9 to 13 years | | | | | | |
| Age | 1 | 1.44 | 4.65[b] | 0.01 | 0.57 | 1.40 |
| Height | 1 | 348.42[a] | 314.12[a] | 307.17[a] | 403.88[a] | 465.10[a] |
| Area | 2 | 6.60[a] | 8.47[a] | 2.80 | 3.55[b] | 2.41 |
| Ethnic differences | 1 | 0.71 | 0.04 | 1.38 | 0.40 | 0.70 |

[a] Significant at 0.01 level.

[b] Significant at 0.05 level.

Table 5.6.7. REGRESSION COEFFICIENTS FOR EFFECTS OF AGE (in liters/yr) AND HEIGHT (in liters/in.) ON $FEV_{0.75}$ BY SEX AND BY AGE GROUP

| Factor | Nov-Dec | Jan | Feb-Mar | Apr | Average |
|---|---|---|---|---|---|
| Males 5 to 8 years | | | | | |
| Age | 0.0549 | 0.0306 | 0.0211 | 0.0725 | 0.0448 |
| Height | 0.0433 | 0.0479 | 0.0486 | 0.0476 | 0.0469 |
| Females 5 to 8 years | | | | | |
| Age | 0.0540 | 0.0356 | 0.0383 | 0.0578 | 0.0465 |
| Height | 0.0399 | 0.0395 | 0.0416 | 0.0475 | 0.0422 |
| Males 9 to 13 years | | | | | |
| Age | 0.0347 | 0.0233 | 0.0159 | 0.0240 | 0.0242 |
| Height | 0.0624 | 0.0598 | 0.0582 | 0.0595 | 0.0600 |
| Females 9 to 13 years | | | | | |
| Age | 0.0159 | 0.0286 | 0.0010 | 0.0098 | 0.0135 |
| Height | 0.0667 | 0.0633 | 0.0647 | 0.0699 | 0.0661 |

For the elementary school population sampled in this study, early childhood exposure to elevated air pollution levels lasting 5 to 10 years appears to have been accompanied by a decrement in ventilatory function. The study also suggests that improvement in ambient air quality may be followed by improved ventilatory function. The study could identify no effect of month-to-month changes in air quality on month-to-month changes in ventilatory function. These changes in ventilatory function may well be attributable in part to a physiologic seasonal variation. The effect of short-term pollution exposures in the present study may have been obscured by a temperature effect, with lower temperatures adversely affecting ventilatory function.

The influence of nine factors must be considered before assessing the implications of the present study for air quality standards: (1) socioeconomic differences, (2) ethnic factors, (3) migration, (4) urban heat island, (5) indoor air pollutants, (6) ambient air pollutants other than those measured, (7) learning, (8) current or recent acute respiratory disease, and (9) the machinery used in testing.

Socioeconomic differences tend to cancel themselves in the present study, and thus could hardly account for the observed differences in lung function. One of the Intermediate communities ranked socioeconomically higher and the other lower than the Low community. Social differences in the study areas were also quite modest when compared to those encountered in mortality studies of the effects of air pollution on health.

Ethnic differences could have biased the study had not the experience of blacks been excluded from the analyses. Blacks have been shown to have lower measured ventilatory performance than whites of comparable age, sex, and height.[10-13] Too few black children from the Intermediate areas were available to achieve the sample size needed for significance testing. Intercommunity differences in ventilatory performance attributable to differences in the proportions of Jewish or Italian surnames were also sought, but none were found (Table 5.6.6).

Migration effects, while not specifically considered, would probably tend to dampen rather than accentuate the observed differences between the Intermediate and Low communities. Children moving to more polluted areas from clean areas are more likely to have better ventilatory function than life-long residents of polluted areas. Similarly, children moving from polluted areas would tend to bring their impaired ventilatory function to the relatively clean communities. Data from other CHESS studies indicated that up to 25 percent of families in the study communities had made such moves across pollution categories.

Lower ambient temperatures seemed generally to be associated with decrements in ventilatory function. However, area temperature differences would also probably tend to dampen rather than accentuate the observed differences attributable to air pollution. The Intermediate communities were integral parts of a compact urban heat island, and winter temperatures were somewhat warmer in New York City than on Long Island. This fact might tend to improve ventilatory performance in the Intermediate communities relative to the Low community.

The effects of indoor air pollutants from cigarette smoking or from domestic use of gas for cooking or space heating could not be quantitated. Though the smoking habits of elementary school children were not documented, there was no particular reason to suspect that their smoking habits would be very different in the Intermediate and Low pollution areas. The prevalence of cigarette smoking among children is probably most strongly influenced by parental smoking habits and by socioeconomic status. In this study, parental smoking habits were documented to be very similar in all three communities. Also, because the Low pollution community was socioeconomically intermediate between the Intermediate I and II communities, differences in smoking habits between Intermediate and Low exposure areas probably would have tended to cancel in this study.

Environmental pollutants other than those measured might also contribute to the observed decrements in ventilatory function. One environmental factor that stands out, distinguishing the Intermediate I exposure community from the other study areas, is its proximity to one of the country's busiest international airports. We were unable to monitor air pollutants peculiar to aircraft emissions. Even with the best existing instrumentation, it would be difficult to monitor the hydrocarbons emitted by aircraft but not by automobile traffic and therefore to specify which pollutants are unique to the area.

Ventilatory performance, like other performance testing, is likely to be enhanced by learning. One would have to postulate significant area differences in the effects of learning to account for the present results, and this would not seem justified. In pulmonary function studies in all CHESS areas, investigators have been consistently impressed by the ease with which even the youngest children learn the FEV procedure. However, there is little doubt that older children grasp the procedure more thoroughly than younger ones do. Thus the results obtained from older children are probably somewhat more reliable than those from younger children.

Concurrent or recent acute respiratory disease might adversely affect ventilatory performance. Excesses in such disorders did in fact occur in the Intermediate communities.[14] However, the study design minimized the effects of acute respiratory illness by excluding symptomatic children from the analysis. One point should be emphasized here: air pollution seems to have played an important role in the excess respiratory disease experience of children in the more polluted communities. Thus, though impaired ventilatory function may be partly attributable to respiratory illness history, this history may in turn be partly attributable to air pollution exposure.

In CHESS studies subsequent to the one reported here, the investigators became aware of a tendency of the pulmonary function indicator to drift a maximum of about 350 ml in the course of a day. The drift was usually downward but occasionally upward. In the present study, the presence and magnitude of drift were never determined. There is no reason to suspect, however, that instrument drift, if present, would have altered one community's results by a different magnitude or in a different direction from another's. (The National Cylinder Gas Pulmonary Function Indicator was replaced in CHESS pulmonary function studies by a waterless dry-seal volume spirometer in 1972.)

In summary, combined covariate effects are likely to have dampened rather than accentuated true differences between the Intermediate and Low communities, or not to have affected these differences at all. We concluded that 9 or more years exposure to annual sulfur dioxide levels of an estimated 131 to 435 $\mu g/m^3$ accompanied by suspended particulate levels of about 75 to 200 $\mu g/m^3$ and suspended sulfate levels of about 5 to 25 $\mu g/m^3$ can be associated with a small but significant impairment in ventilatory function that persists even as Primary Ambient Air Quality Standards for these pollutants are achieved. Furthermore, though it is unlikely, one cannot be certain that lower annual levels of sulfur dioxide (66 to 76 $\mu g/m^3$) accompanied by lower annual levels of suspended sulfates (13 $\mu g/m^3$) and suspended particulates (61 to 74 $\mu g/m^3$) do not facilitate the persistence of previously induced impairments in ventilatory function. Confirmation of the adequacy of present air quality standards must await the passage of time and the necessary retesting of ventilatory function. Finally, time may well prove that more attention should be devoted to the effects of suspended sulfates.

## SUMMARY

Ventilatory function ($FEV_{0.75}$) tests were performed by 2364 New York metropolitan elementary school children four times during the 1970-1971 school year. Older white children, aged 9 to 13 years, from a relatively unpolluted suburban fringe community on Long Island were found to have significantly better ventilatory function than children from two more polluted urban communities. This difference existed in the face of recent improvements in air quality. Older children exposed for more than 8 years to annual average sulfur dioxide levels estimated at 131 to 435 $\mu g/m^3$, accompanied by levels of suspended particulates estimated at 75 to 200 $\mu g/m^3$ and suspended sulfates estimated at 5 to 25 $\mu g/m^3$ showed significant decreases in $FEV_{0.75}$, but younger children, age 5 to 8 years, did not. The effects of age, height, socioeconomic differences, ethnic factors, migration, ambient temperature, exposure to indoor air pollutants and to pollutants not monitored, and concurrent acute respiratory disease were all considered.

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# CHAPTER 6

# CINCINNATI STUDY

# 6.1 VENTILATORY FUNCTION IN SCHOOL CHILDREN: 1967-1968 TESTING IN CINCINNATI NEIGHBORHOODS

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## INTRODUCTION

Prolonged exposure to air pollution in the early years of life can produce adverse changes in pulmonary function of children. Holland et al.[1] detected reduced function in urban children attending schools in the more polluted areas of Kent, England. Children residing in a heavily polluted metropolitan area of Japan had significantly lower forced-expiratory-volume measurements than children in a rural area; these differences were greatest during winter months when urban pollutant levels were highest.[2] Similar results have not been reported in United States cities having only moderately elevated concentrations of particulate and sulfur oxide pollution. The purpose of this study was to investigate the influence of temporal and geographic variation in urban air pollution on ventilatory performance of school children living in a moderately large metropolitan area of the United States. Possible effects of both acute and chronic exposures were sought.

## MATERIALS AND METHODS

The study was designed to include a pair of public elementary schools in each of six neighborhoods differing in socioeconomic level, race, or pollution exposure, as shown in Table 6.1.1. The six schools in polluted neighborhoods were located in the main industrial valley of central metropolitan Cincinnati and were matched for socioeconomic and racial characteristics with six schools in a nonindustrial river valley on the east side of the metropolitan area. Visual estimates of home market values were used to gauge the general socioeconomic level of the neighborhood. These estimates were substantiated by questionnaires administered to households of participating children at the end of the study. One school had approximately equal numbers of black and white children and is listed twice in Table 6.1.1 as Schools 8 and 12. All children in one or two classrooms of the second grade of the elementary schools were asked to participate in the study to achieve sample sizes of 60 to 75 children in each of the six study sectors.

**Table 6.1.1. COMPOSITION OF STUDY SECTORS ACCORDING TO SOCIOECONOMIC-RACIAL AND EXPOSURE CATEGORIES**

| Socioeconomic and racial category | Exposure category | |
|---|---|---|
| | Clean | Polluted |
| Upper-middle white | School 1<br>School 2 | School 3<br>School 4[a] |
| Lower-middle white | School 5<br>School 6 | School 7<br>School 8[b] |
| Lower-middle black | School 9<br>School 10 | School 11<br>School 12[b] |

[a] This school was reassigned to the clean exposure category on the basis of pollutant data obtained during the 8-month study.

[b] Schools 8 and 12 are, respectively, the white and black portions of the same school.

Ventilatory performance, as measured by a 0.75-second forced-expiratory volume ($FEV_{0.75}$) on a Stead-Wells spirometer, was obtained 12 times from each child: once weekly in the months of November 1967 and February and May 1968. Tests were administered on Tuesday and Wednesday mornings by six teams, which were systematically rotated among schools. The best of two satisfactory forced-expiratory maneuvers obtained on each test day was used for all computations. Criteria for a "satisfactory" maneuver were: (1) full inspiration before exhaling, (2) maximum, uninterrupted exhalation, and (3) no leakage of air from around the mouthpiece. Each child was asked whether he currently had a cold, cough, or sore throat on the test day. Standing heights were measured to the lower half inch once monthly, and a linear regression of $FEV_{0.75}$ on height was computed to adjust for area differences in height of children.

In the final month of study, experienced Census Bureau interviewers visited homes and administered a health questionnaire to the mother of each participating child. Questions were asked about the child's past history of illness, parental income, education, occupation, and duration of residence at current address.

Air monitoring stations were placed in locations within three blocks of each school to provide samples representative of the air quality in the neighborhood served by the school. Twenty-four-hour integrated samples of total suspended particulates, suspended sulfates and nitrates, and sulfur dioxide were collected daily during the 8 months of the study at the monitoring station adjacent to each school. At one school in each of the six study sectors, both outdoor and indoor soiling index and sulfur dioxide were monitored. During the 12 hours preceding pulmonary function testing, 4-hour sequential samples of soiling index and sulfur dioxide were obtained at these schools. Daily measurements of 24-hour total suspended particulates, suspended sulfates, and suspended nitrates were collected with high-volume air samplers designed to capture particles less than 90 microns in diameter.[3] Soiling-index samples were collected on a sequential tape sampler with a 4-hour sampling cycle at a flow rate of 15 cubic feet per hour.[4] Sulfur dioxide was measured by bubbling air through a solution of tetrachloromercurate and analyzed by the colorimetric method of West and Gaeke.[5]

## RESULTS

Pollutant concentrations at School 4 (polluted, upper-middle class, white sector) were consistently lower than expected and were similar to schools in the clean areas of the study. School 4 was therefore reassigned to the clean, upper-middle class, white sector, leaving only one school in the polluted category of this socioeconomic level.

Concentrations of the various pollutants averaged over the 7 months of study are presented for each study sector in Figure 6.1.1 and for each school in Table 6.1.2. Seasonal averages for total suspended

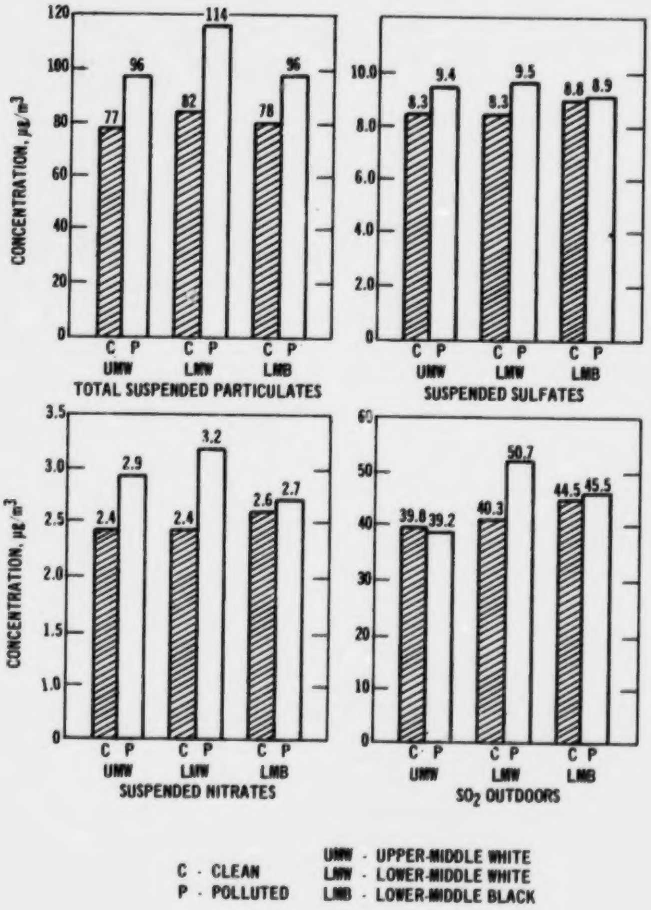

Figure 6.1.1. Pollutant concentration, arithmetic averages over entire 7 months of study within each study sector, Cincinnati, 1967-1968.

### Table 6.1.2. ARITHMETIC AVERAGES OF POLLUTANT CONCENTRATIONS MEASURED OVER ENTIRE 7 MONTHS OF STUDY FOR EACH SCHOOL, OCTOBER 24, 1967 to MAY 22, 1968
(n = 211 sampling days)

| Stratum | School | Suspended particulates, $\mu g/m^3$ | | | Outdoor | | Indoor | |
|---|---|---|---|---|---|---|---|---|
| | | Total | Sulfate | Nitrate | Soiling, COH | $SO_2$, $\mu g/m^3$ | Soiling, COH | $SO_2$, $\mu g/m^3$ |
| Clean UMW | 1 | 61 | 8.2 | 2.2 | | 37 | | |
| | 2 | 85 | 7.7 | 2.4 | 0.87 | 39 | 0.72 | 10 |
| | 4 | 85 | 9.1 | 2.7 | | 47 | | 5 |
| Polluted UMW | 3 | 96 | 9.4 | 2.9 | 1.05 | 39 | 0.77 | 16 |
| Clean LMW | 5 | 88 | 8.2 | 2.4 | 0.90 | 44 | 0.92 | 15 |
| | 6 | 76 | 8.4 | 2.3 | | 37 | | |
| Polluted LMW | 7 | 133 | 10.1 | 3.6 | 1.35 | 57 | 1.25 | 21 |
| | 8 | 96 | 8.9 | 2.8 | | 44 | | |
| Clean LMB | 9 | 78 | 8.9 | 2.6 | | 50 | | |
| | 10 | 78 | 8.7 | 2.6 | 0.98 | 39 | 0.85 | 16 |
| Polluted LMB | 11 | 97 | 8.9 | 2.6 | 1.04 | 47 | 0.91 | 6 |
| | 12 | 96 | 8.9 | 2.8 | | 44 | | |

particulates ranged from 61 to 92 $\mu g/m^3$ in clean neighborhoods, and from 76 to 131 $\mu g/m^3$ in polluted neighborhoods, a relative difference of 33 percent. During the winter months (December-February) suspended particulate concentrations increased by 25 to 30 percent in all areas (Table 6.1.3), but relative differences between clean and polluted neighborhoods remained the same.[6]

A total of 394 second-grade children, representing 93 percent of enrollment in selected classrooms, participated in the study during all 3 months of pulmonary function testing. As shown in Table 6.1.4, sample sizes ranged from 44 to 99 children in the various study sectors.

Within the same socioeconomic category, residents of clean and polluted neighborhoods were similarly distributed with respect to educational achievement of the head of household (Figure 6.1.2). Differences between socioeconomic categories were large. Income and occupation of father, market value of home, monthly rent and number of persons per bedroom showed the same area distribution patterns as education.

Analysis of $FEV_{0.75}$ results was performed only on students who participated in each of the 3 months of pulmonary function testing. From 5 to 13 percent of children in the various study sectors had a history of asthma or bronchitis; these children, numbering 46 in all, were eliminated from the analysis, reducing the total sample size to 348.

### Table 6.1.3. SEASONAL VARIATION OF TOTAL SUSPENDED PARTICULATE AND OUTDOOR 24-HOUR AVERAGE SULFUR DIOXIDE WITHIN EXPOSURE SECTORS
($\mu g/m^3$)

| Season and socioeconomic-racial category | Exposure category | | | |
|---|---|---|---|---|
| | Clean | | Polluted | |
| | TSP | $SO_2$ | TSP | $SO_2$ |
| Fall (Oct.-Nov.) | | | | |
| UMW | 62 | 29 | 79 | 24 |
| LMW | 66 | 29 | 88 | 39 |
| LMB | 60 | 29 | 76 | 37 |
| Winter (Dec.-Feb.) | | | | |
| UMW | 86 | 58 | 107 | 58 |
| LMW | 92 | 60 | 131 | 73 |
| LMB | 86 | 66 | 106 | 68 |
| Spring (Mar.-May) | | | | |
| UMW | 75 | 26 | 92 | 29 |
| LMW | 82 | 26 | 111 | 34 |
| LMB | 80 | 31 | 98 | 29 |

Weekly $FEV_{0.75}$ values for each sector were adjusted to a standing height of 50 inches by means of the equation:

$$\text{adjusted FEV}_{0.75} = \text{average FEV}_{0.75} + b(50 - \text{average height})$$

A sex-specific regression coefficient, b, was calculated by fitting a least-squares-regression equation to observed $FEV_{0.75}$ values plotted against standing height for each sex. Sex-specific, height-adjusted $FEV_{0.75}$ values were thus computed for each week. A combined value for both sexes was derived by giving equal weight to values for males and females. These combined values are presented in Figure 6.1.3 for each of the 12 weeks of testing. Relatively low $FEV_{0.75}$ values were independently associated with three factors: (1) black children, (2) residence in polluted area, and (3) the month of February. Among white children, the contrast between $FEV_{0.75}$ values of children in polluted and clean neighborhoods was greatest during February. $FEV_{0.75}$ values of black children in polluted areas were not consistently different than values of black children in clean areas.

**Table 6.1.4. NUMBER OF CHILDREN TESTED DURING ALL 3 MONTHS OF STUDY WITHIN EACH SECTOR**

| Socioeconomic-racial category | Exposure category | | |
|---|---|---|---|
| | Clean | Polluted | Total |
| Upper-middle white | 99 | 44 | 143 |
| Lower-middle white | 67 | 53 | 120 |
| Lower-middle black | 62 | 69 | 131 |
| Total | 228 | 166 | 394 |

A multivariate analysis of variance model was used to test the significance of race, socioeconomic contrast between white children, residence in polluted or clean area, standing height, and sex on $FEV_{0.75}$ for the 3 test months (Table 6.1.5). This analysis confirmed the highly significant ($p < 0.01$) effects of height, sex, and race on $FEV_{0.75}$. Among white children, higher socioeconomic level was associated with higher $FEV_{0.75}$ performance. High suspended particulate levels were consistently associated with significantlv lower $FEV_{0.75}$ results. Among pollutants, however, suspended sulfates exerted the strongest influence on lung function of the children. Suspended nitrates also exerted a significant effect on $FEV_{0.75}$. The relative effects of race, sex, and suspended sulfate and nitrate exposure on $FEV_{0.75}$ performance are illustrated in Figure 6.1.4. $FEV_{0.75}$ values have been adjusted for differences in all factors other than those contrasted in that set. Of the four factors, suspended sulfate exposure had the largest independent effect on lung function. On the average, adjusted $FEV_{0.75}$ results for children in polluted areas were 17.4 percent below those for children in clean areas. Lung function of black children was 9.3 percent below that of white children, while results for girls were 6.7 percent below those for boys. The pollution effect was somewhat diminished by the fact that area differences in pollution had no effect on the performance of black children.

Indoor and outdoor sulfur dioxide, soiling index, and suspended particulate levels measured over the 24-hour or 4-hour period directly preceding pulmonary function tests did not consistently correlate with $FEV_{0.75}$ values. Ventilatory performance of children thus did not appear to be acutely affected by variations in pollutant levels on the day of the test.

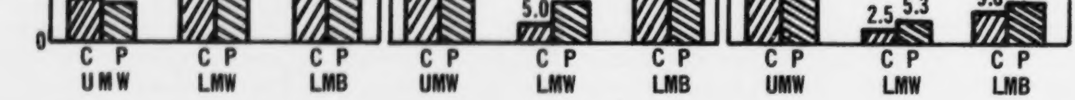

Figure 6.1.2. Educational achievement of fathers within each study sector, Cincinnati, 1967-1968.

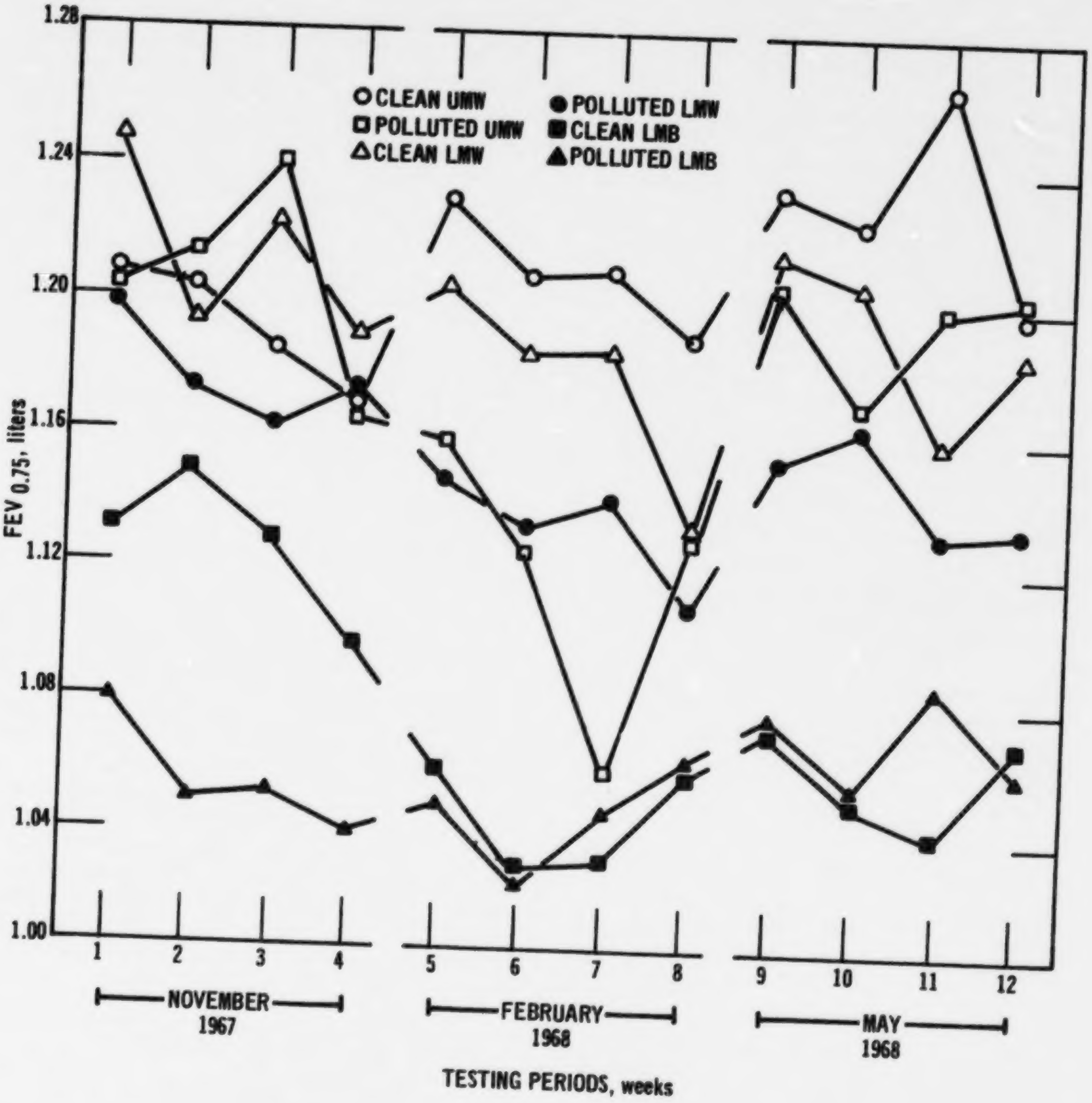

Figure 6.1.3. Height-adjusted $FEV_{0.75}$ during each of 12 weeks of testing for each study sector, Cincinnati, 1967-1968.

$FEV_{0.75}$ results obtained from students admitting to a cold, cough, or sore throat were compared with results from the same children when they denied these symptoms. No consistent difference in $FEV_{0.75}$ values, as tested by paired t-tests, was found.

Average concentrations of total suspended particulates in Cincinnati are available from the National Air Surveillance Network station over the period 1957 to 1968.[7-13] These years cover more than the life span of the 7- to 9-year-old children participating in this study. At the National Air Surveillance Network

### Table 6.1.5. EFFECTS OF AIR POLLUTION, SOCIOECONOMIC-RACIAL CATEGORY, SEX, STANDING HEIGHT, AND MONTH OF TEST ON $FEV_{0.75}$: TESTS OF SIGNIFICANCE BY MULTIVARIATE ANALYSIS OF VARIANCE

| Factor | Regression coefficients[a] | | | Significance test | |
|---|---|---|---|---|---|
| | Nov. | Feb. | May | F | p |
| Suspended particulates | | | | | |
| Sulfates | -0.0145 | -0.0621 | -0.0511 | 8.79 | $<0.01$ |
| Nitrates | -0.0045 | -0.0626 | -0.0420 | 3.56 | $<0.05$ |
| Total particulates | -0.0000 | -0.0008 | -0.0005 | 1.08 | NS[b] |
| UMW versus LMW | 0.0265 | 0.0314 | 0.0548 | 6.07 | $<0.01$ |
| Race | -0.0702 | -0.0687 | -0.0741 | 11.84 | $<0.01$ |
| Sex | 0.0300 | 0.0399 | 0.0388 | 6.84 | $<0.01$ |
| Standing height | 0.0553 | 0.0509 | 0.0528 | 66.56 | $<0.01$ |
| Overall mean $FEV_{0.75}$ | 1.1592 | 1.1500 | 1.1906 | | |

[a] Regression coefficients for socioeconomic level, race, sex, standing height, and mean $FEV_{0.75}$ values are given from the multivariate analysis using sulfates alone as the pollution variable. Regression coefficients changed very little when the analysis was reported for the other pollution variables.

[b] NS—not significant.

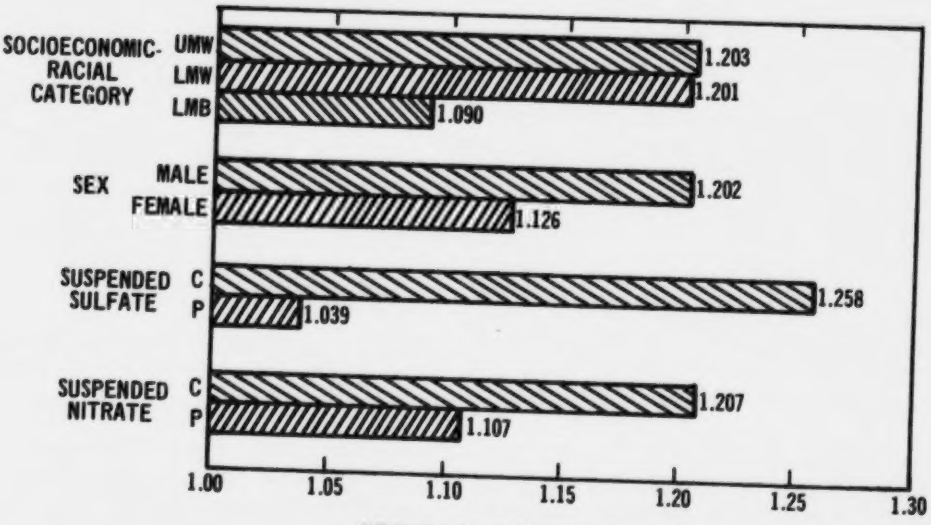

Figure 6.1.4. Relative influence of socioeconomic-racial difference, sex, and air pollution on average $FEV_{0.75}$ performance of school children, Cincinnati, 1967-1968.

station, total suspended particulate concentrations averaged 125 $\mu g/m^3$ (geometric mean) over the 1957 to 1961 period, representing the infant years of participating children. During early childhood years, from 1962 to 1966, these concentrations averaged 129 $\mu g/m^3$ (geometric mean). During the years of this study (1967-1968), however, total suspended particulate concentrations had fallen to an average of 105 $\mu g/m^3$ (geometric mean). Thus, suspended particulate concentrations at the National Air Surveillance Network station were approximately 20 percent higher during the early childhood years than during the years of this study. If diminution in pulmonary function were a response to early childhood exposures rather than to recent pollution exposures, the observed effect on ventilatory performance would be related to total suspended particulate levels estimated to be approximately 20 percent higher than those reported during the study in high exposure neighborhoods. That is, total suspended particulate concentrations are estimated to have been in the range of 115 to 137 $\mu g/m^3$ (arithmetic mean) during early childhood years of the participants from high exposure neighborhoods. A similar time trend for suspended sulfates in high exposure neighborhoods would raise the estimate of exposure associated with diminished lung function in neighborhoods of white children from observed values between 8.9 and 10.1 $\mu g/m^3$ to values between 10.7 and 12.1 $\mu g/m^3$. For suspended nitrates, estimates of potentially detrimental exposure would be raised from 2.8 and 3.6 $\mu g/m^3$ to 3.4 and 4.3 $\mu g/m^3$. These worst-case and least-case estimates are given for pollutant exposures most strongly associated with diminished lung function of the children participating in this study.

## DISCUSSION

This study showed significant area differences in ventilatory performance of second-grade school children. Area differences were associated with both race and chronic exposure to moderate levels of suspended sulfates and nitrates in the air.

Densen and his coworkers[14,15] also noted lower pulmonary function and lower smoking-specific chronic respiratory symptom rates among adult black New York City postal workers compared with whites in the same occupation. The strong racial difference in ventilatory function among school children has also been previously reported.[16-19] The ventilatory performance of black children was variously reported as 7 to 15 percent[18,19] below that of white children, and differences persisted when similar socioeconomic groups of the two races were compared[17] or when adjustments were made for differences in stem height and chest expansion.[18] One possible explanation for the relatively low $FEV_{0.75}$ values of the black children in our study was their response, conditioned by racial attitudes prevalent in their early years, to the all-white teams administering the test.

Of the three measured components of suspended particulate matter (total, sulfates, and nitrates), suspended sulfate levels had the strongest quantitative association with area differences in $FEV_{0.75}$ (Figure 6.1.4). Area differences in suspended sulfates between clean and polluted neighborhoods were largest among UMW and LMW sectors, but almost nonexistent among LMB sectors (Figure 6.1.1). The same gradients of exposure existed for suspended nitrates. Decrements in $FEV_{0.75}$ performance paralleled the gradients of exposure to suspended sulfates and nitrates. Clean and polluted black sectors, however, showed relatively large differences in total suspended particulates—a difference not reflected in $FEV_{0.75}$ performance. Thus, for establishing appropriate dose-response relationships, quantitative differences in pollutant exposure of UMW and LMW study sectors are the only useful considerations. That is, LMB areas showed no area difference in lung function that was consistent with differences in pollution exposure. Mean pollutant exposures in white sectors with low and high exposure, respectively, were as follows (values given are arithmetic means in clean versus polluted sectors): 80 vs 105 $\mu g/m^3$ for total suspended particulates, 8.3 vs 9.5 $\mu g/m^3$ for suspended sulfates, and 2.4 vs 3.1 $\mu g/m^3$ for suspended nitrates (Figure 6.1.1).

These levels were probably about 20 percent higher during 7 to 9 years of exposure preceding this study for children who were lifetime residents of Cincinnati. The fact that lung function of children in high-exposure neighborhoods improved in spring when air quality also improved suggests that current pollutant exposures, in addition to chronic exposures, may exert an effect on ventilatory performance. Unfortunately, little information on lung function values is as yet available to test this hypothesis. The springtime improvement in $FEV_{0.75}$ may also, in part, reflect a physiologic seasonal variation in ventilatory function. The results of this study support the hypothesis that chronic exposure to these relatively low levels of suspended particulates was associated with significantly lower ventilatory performance of second-grade school children. These effects occurred in the presence of low concentrations of sulfur dioxide (less than 52 $\mu g/m^3$) in all areas and constitute one of the few cases where health effects can be attributed to particulate pollution independently of atmospheric levels of gaseous sulfur dioxide.

As shown in Figure 6.1.3, the magnitude of differences in ventilatory performance of children in clean and polluted schools in the two white socioeconomic categories was greatest during February, when both low outdoor temperatures and high concentrations of air pollutants were experienced. Cold temperatures alone did not significantly affect the $FEV_{0.75}$ results of children in the clean areas, and ventilatory performance of children was not affected by acute exposure to pollutants on the day of the test. An interaction between cold weather and elevated pollutant concentrations in polluted neighborhoods alone is a possible explanation for these results. The data in Figure 6.1.3 suggest that the impairment of ventilatory performance among children of this age group was not irreversible, for during May, $FEV_{0.75}$ differences between white children in clean and polluted neighborhoods were diminished. In retrospect, it would have been highly desirable to have repeated ventilatory performance tests on these same children in November of 1968, to determine whether differences in $FEV_{0.75}$ values again became very slight or disappeared altogether.

Other investigators have reported an association between ventilatory performance of school children and air pollution exposure. On comparing pulmonary function of 10- to 11-year-old school children in a heavily polluted area and a clean area of Osaka, Japan, during each of 12 successive months, Watanabe et al.[20] found peak flow rates to be associated with geographic and temporal differences in exposure to sulfur dioxide and particulate matter. Area differences in peak flow rates were greater during winter months when pollutant concentrations were highest in the polluted area. Total vital-capacity measurements did not show this relationship with area; however, no corrections were made for observed differences in height between the children of the two areas. In a similar comparison of 10- and 11-year-old children in an industrialized area and a rural area of Kawasaki, Japan, Toyoma[2] reported diminished peak flow rates and one-half-second forced-expiratory-volume measurements in children of the highly polluted area. Area differences were again greatest during the season of highest pollution; however, in Kawasaki highest concentrations of pollution occurred during the warmer months of April through August. Thus pollution exposure exerted its effect independently of cold weather. Sulfur dioxide and particulate matter levels were simultaneously elevated, preventing discrimination of the effects due to each pollutant alone.

Petrilli et al.[21] reported that peak flow and mid-maximal expiratory flow rates of adolescents in a relatively polluted industrial area of Genoa, Italy, were lower than tests obtained from adolescents in a residential sector of the same city. Slight area differences in age and height were observed. but pulmonary function results were not adjusted for these differences, nor were socioeconomic characteristics of the two areas described.

Anderson and Kinnis,[22] comparing first-grade school children in a British Columbia kraft pulp mill town with an adjacent clean community, found age- and height-adjusted peak flow rates of male children in the mill town to be significantly lower than male rates in the control town; these differences were not found among female children. No air pollution measurements were made in this study.

Five- and six-year-old children living in the most polluted of four sectors of Sheffield, England, had significantly lower height-adjusted $FEV_{0.75}$ values than children in the other sectors.[23] The high-exposure sector had high concentrations of both sulfur dioxide and suspended particulates. Pulmonary function measurements were made during summer months when pollution levels were relatively low and acute respiratory infections were infrequent.

Holland et al.[1] demonstrated significantly lower peak expiratory flow rates, after adjusting for the effect of social class, family size, and past history of respiratory illness, in urban school children of Kent, England, living in a moderately polluted residential area.

McMillan et al.[24] failed to find an effect of photochemical oxidant pollution on peak flow rates of third-grade school children living in two suburbs of Los Angeles. Substantial area differences in ethnic composition and family stability were present, however, and may have influenced peak flow rates in a direction opposite to that of air pollution.

These studies point to a consistent relationship between impaired ventilatory performance of children 5 to 10 years of age and exposure to sulfur dioxide in combination with particulate matter. Impairment was not associated with overt clinical disease and could be detected only by comparison with performance of children in control communities matched for socioeconomic characteristics. Important covariates of ventilatory performance in children, including age, height, socioeconomic level, and history of respiratory disease, were taken into account in some of these studies. The influences of cigarette smoking and of occupational exposures to respiratory irritants were minimized by selection of elementary school children for study. Performance of children in the polluted communities generally improved during seasons of low pollution, but did not attain the level of their counterparts in clean areas, suggesting a chronic but still reversible state of functional impairment.

The present study confirms the findings of those from other countries. Significantly lower ventilatory performance, adjusted for height, socioeconomic level, and race, was present in elementary school children exposed to relatively moderate levels of particulate matter, ranging from 76 to 131 $\mu g/m^3$ compared to children exposed to 61 to 92 $\mu g/m^3$. Lower ventilatory performance was most highly associated with exposures to suspended sulfate concentrations of 8.9 to 10.1 $\mu g/m^3$ monitored during the 7 months of the study, and with suspended sulfate concentrations of 10.7 to 12.1 $\mu g/m^3$ estimated for the lifetime exposure of children who always lived in the same high exposure neighborhoods in Cincinnati. This effect appears to have occurred independently of atmospheric sulfur dioxide, levels of which were low (less than 52 $\mu g/m^3$) in all areas. Absence of an air pollution effect among blacks is difficult to evaluate in view of the very low performance of black children relative to the white and the possibility that the low performance may have been a culturally determined response of these children to the white interviewers.

## SUMMARY

Ventilatory performance, measured as $FEV_{0.75}$, of white children residing in a moderately polluted industrial valley of Cincinnati was significantly lower than the performance of white children living in a clean area of the metropolitan region. Areas were matched for socioeconomic and racial characteristics. Tests were performed once weekly for a month during three different seasons of the school year. Area differences in test results were greatest during the cold winter month when pollution levels were highest in all sectors. Socioeconomic level had an influence on the ventilatory performance of white children, but black children of both exposure areas had the lowest test results of all groups. Pollution exposure had no consistent area effect on the performance of the black children. In none of the study sectors were test results significantly associated with pollution concentrations during the 24 or 4 hours immediately preceding the tests.

Concentrations of total suspended particulates, suspended sulfates and nitrates, soiling index, and sulfur dioxide were measured daily at monitoring stations established adjacent to all schools in the six study sectors. Indoor and outdoor concentrations were monitored at one school in each study sector. Sulfur dioxide levels were low—the 7-month arithmetic mean was less than 52 $\mu g/m^3$—in all sectors. The largest differences in area exposure were in the concentrations of suspended particulates. Levels of total suspended particulates were 76 to 131 $\mu g/m^3$ in polluted sectors and 61 to 92 $\mu g/m^3$ in clean sectors, a relative difference of 32 percent. In the polluted sectors, the average suspended sulfate level was 9.5 $\mu g/m^3$, compared to 8.3 $\mu g/m^3$ in the clean white sectors, a relative difference of 13 percent. In the polluted white sectors, the average suspended nitrate level was 3.1 $\mu g/m^3$, compared to 2.4 $\mu g/m^3$ in the clean white sectors, a relative difference of 23 percent. Since sulfur dioxide levels were low in all areas, observed area differences in $FEV_{0.75}$ were probably attributable to differences either in total suspended particulate, suspended sulfate, or nitrate exposure. Black children were exposed to substantially different levels of total suspended particulate, yet showed no appreciable differences in ventilatory performance. Because ventilatory performance varied most strongly with exposure to suspended sulfates in all racial and socioeconomic groups, the observed area effect on $FEV_{0.75}$ was probably attributable mostly to suspended sulfates. However, the socioeconomic-racial exposure gradients of suspended nitrates were identical to the gradients of suspended sulfates. Clearly, the relative effects of total suspended particulates, suspended sulfates, and suspended nitrates need further elucidation. The possibility that suspended sulfates and nitrates exert a combined detrimental effect on ventilatory function is of particular concern.

This study substantiated the evidence from several other countries that pulmonary function of young elementary school children is adversely affected by chronic moderate levels of air pollution exposure.

## ACKNOWLEDGMENT

The authors gratefully acknowledge the services of Miss Kathryn McClain who carefully measured all spirograms and helped to prepare tabulations for this paper.

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# CHAPTER 7
# SUMMARY AND CONCLUSIONS

# 7.1 HEALTH CONSEQUENCES OF SULFUR OXIDES: SUMMARY AND CONCLUSIONS BASED UPON CHESS STUDIES OF 1970-1971

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## INTRODUCTION

Epidemiologic studies of the U. S. Environmental Protection Agency's Community Health and Environmental Surveillance System (CHESS) program are designed to develop dose-response information relating short-term and long-term pollutant exposures to adverse health effects.[1] This paper summarizes effects associated with sulfur oxide exposures in CHESS communities in New York and the Salt Lake Basin during studies conducted in 1970-1971. In addition, studies conducted in Idaho-Montana (Rocky Mountain studies), Chicago, and Cincinnati, in which health indicators similar to those used in CHESS were employed, are included. The individual research reports on which this study is based often suggested pollutant-disease associations but left a number of problems unanswered. These problems include (1) the relative contribution of various air pollutants, especially sulfur dioxide ($SO_2$), total suspended particulates (TSP), and suspended sulfates (SS), to observed disease frequencies; (2) the importance of intervening influences, or covariates, such as occupational exposures, socioeconomic status, residential mobility, and cigarette smoking; (3) the association between chronic disease prevalence and current versus past pollutant exposures; and (4) the precise pollutant threshold for excess disease in exposed communities.

Obviously, epidemiologic studies alone cannot resolve any one of the above problems. The findings summarized in this paper must be substantiated by replicated observations in different years and under different circumstances. Well controlled human and animal studies are required to isolate several of the important intervening variables that are inherent to studies of free living populations, and to elucidate the precise nature of the pollutant-disease relationship. Hence, the conclusions put forth at this time cannot be definitive, but are offered in the sense of developing more refined quantitative and scientific hypotheses concerning pollutant-health effect associations in a real-life environment. In the CHESS program, we are repeating our observations, using essentially the same health indicators in the same (and more) communities.[1] These results will provide one form of data verification required for scientifically defensible air quality standards.

In relating observed health effects to possible pollutant thresholds, wherever practical and possible, three threshold estimates were provided: a worst case estimate, which attributes an observed adverse health effect to the *lowest pollution exposure* suggested by the epidemiologic studies after considering only the strongest and most established covariates; a least case estimate, which attributes an observed adverse health effect to the *highest pollution exposure* level suggested by the epidemiologic studies after considering effects of all covariates; and a best judgment estimate based upon a *synthesis* of several studies. The best judgment estimate duly considers interactions between pollutants and is at times based upon special analyses that were necessary when individual studies raised questions regarding interactions involving pollutants or intervening variables.

## RESPONSES TO LONG-TERM POLLUTANT EXPOSURES

### Summary of Chronic Respiratory Disease Studies

Chronic bronchitis prevalence rates observed in four CHESS areas are given in Table 7.1.1.[2-5] (For the studies summarized, chronic bronchitis was characterized by the presence of cough and phlegm on most days for at least 3 months each year.) In each of the four studies, a very consistent pattern of excess chronic bronchitis was found among residents of more polluted communities. In each case, these differences were statistically significant. Mean respiratory symptom scores, which take into account less severe as well as more classical chronic respiratory symptoms, are tabulated for each CHESS area in Table 7.1.2. Symptom scores substantiate the consistent pattern of excess respiratory symptoms among participants from more polluted neighborhoods. (As indicated by the difference in community designations, pollutant gradients in the several CHESS areas do not represent equal class intervals. Pollutant ranges associated with adverse health effects are presented later.)

In the Salt Lake, Rocky Mountain, and New York surveys[2,3,5] where parents of school children were studied, several consistent findings were observed. For both smokers and nonsmokers, there was a male excess of chronic respiratory disease, whether defined in terms of bronchitis prevalence or of symptom scores. Chronic bronchitis rates and symptom scores were higher among male and female smokers. Finally, male and female nonsmokers, exsmokers, and current smokers from more polluted areas had higher chronic

### Table 7.1.1. SMOKING- AND SEX-SPECIFIC CHRONIC BRONCHITIS PREVALENCE RATES BY COMMUNITY IN FOUR CHESS AREAS

| CHESS area and community | Percent chronic bronchitis | | | | | |
|---|---|---|---|---|---|---|
| | Nonsmoker | | Exsmoker | | Smoker | |
| | Male | Female | Male | Female | Male | Female |
| Salt Lake Basin | | | | | | |
| Low | 3.0 | 2.3 | 2.6 | 5.3 | 19.9 | 17.8 |
| Intermediate I | 3.6 | 2.0 | 3.4 | 4.0 | 18.6 | 14.7 |
| Intermediate II | 2.3 | 4.7 | 5.4 | 7.0 | 20.1 | 15.3 |
| High | 6.8 | 5.2 | 6.0 | 7.1 | 26.8 | 22.2 |
| Rocky Mountain | | | | | | |
| Pooled Low | 1.25 | 1.08 | 1.45 | 3.12 | 17.05 | 11.78 |
| Pooled High | 3.47 | 2.54 | 4.82 | 2.80 | 18.63 | 12.88 |
| Chicago | | | | | | |
| Black | | | | | | |
| Outstate (Low) | 8.8 | | | | 8.8 | |
| Suburban (High II) | 7.8 | | | | 12.7 | |
| Urban (High I) | 9.5 | | | | 13.0 | |
| White | | | | | | |
| Outstate (Low) | 4.2 | | | | 17.6 | |
| Suburban (High II) | 5.4 | | | | 18.8 | |
| Urban (High I) | 5.4 | | | | 17.8 | |
| New York | | | | | | |
| Low | 4.6 | 2.0 | 13.9 | 3.8 | 13.9 | 13.9 |
| Intermediate I | 18.0 | 7.5 | 18.0 | 9.0 | 21.3 | 19.8 |
| Intermediate II | 14.2 | 4.9 | 18.7 | 4.5 | 22.1 | 16.6 |

bronchitis rates and symptom scores than those from less polluted areas.

Despite considerable variation in the population characteristics and pollutant exposures of the above three CHESS areas, the relative contribution of cigarette smoking alone was greater than the effect of the air pollution gradient (Table 7.1.3), with the exception of males in New York. Among males in the Salt Lake and Rocky Mountain CHESS areas, and among all females, air pollution alone was associated with an excess bronchitis rate (when compared with nonsmokers of less polluted neighborhoods) ranging from 1.5 to 3.8 percent. Among New York City males, this excess was 11.3 percent – an unusually high figure requiring verification in subsequent study years. Cigarette smoking alone accounted for an excess bronchitis rate of 9.3 to 16.6 percent in the three CHESS areas. Thus, the relative contribution of air pollution alone ranged from one-third to one-seventh as strong as that of cigarette smoking as a determinant of chronic bronchitis prevalence in communities (with the exception of males in New York, where air pollution appeared to make a slightly larger contribution than smoking – a finding difficult to accept in the light of other evidence). The range of observed differences in the relative contributions of smoking and pollution is not surprising in view of the quantitative and qualitative differences in pollution profiles of the communities studied as well as the community differences in smoking patterns. The sum of the evidence suggests that, while personal cigarette smoking is the largest determinant of bronchitis prevalence among parents of school children, air pollution itself is a significant and consistent contributing

Table 7.1.2. MEAN RESPIRATORY SYMPTOM SCORES FOR ALL STAGES OF CHRONIC RESPIRATORY SYMPTOMS IN FOUR CHESS AREAS

| CHESS area and community | Mean symptom score | | | | | |
|---|---|---|---|---|---|---|
| | Nonsmoker | | Exsmoker | | Smoker | |
| | Male | Female | Male | Female | Male | Female |
| Salt Lake Basin | | | | | | |
| Low | 1.54 | 1.36 | 1.48 | 1.65 | 2.73 | 2.57 |
| Intermediate I | 1.56 | 1.37 | 1.54 | 1.79 | 2.88 | 2.53 |
| Intermediate II | 1.48 | 1.55 | 1.82 | 1.88 | 2.99 | 2.44 |
| High | 1.94 | 1.73 | 1.87 | 2.02 | 3.32 | 2.98 |
| Rocky Mountain | | | | | | |
| Pooled Low | 1.33 | 1.23 | 1.41 | 1.30 | 2.65 | 2.20 |
| Pooled High | 1.38 | 1.29 | 1.58 | 1.45 | 2.72 | 2.34 |
| Chicago | | | | | | |
| Black | | | | | | |
| Outstate (Low) | 2.10 | | | | 2.47 | |
| Suburban (High II) | 2.24 | | | | 2.61 | |
| Urban (High I) | 2.34 | | | | 2.71 | |
| White | | | | | | |
| Outstate (Low) | 1.76 | | | | 2.90 | |
| Suburban (High II) | 1.82 | | | | 2.93 | |
| Urban (High I) | 1.84 | | | | 2.91 | |
| New York | | | | | | |
| Low | 1.81 | 1.29 | 2.05 | 1.37 | 2.48 | 2.30 |
| Intermediate I | 2.41 | 1.76 | 2.56 | 2.00 | 2.76 | 2.68 |
| Intermediate II | 2.35 | 1.61 | 2.51 | 1.72 | 2.76 | 2.53 |

factor, leading to increased bronchitis rates in nonsmokers as well as smokers from polluted communities.

Among young white military recruits studied in the Chicago area,[4] air pollution was associated with a considerably smaller excess in bronchitis rates (Table 7.1.3) than was found in the other CHESS areas, and the contribution of air pollution was relatively much less than that of cigarette smoking. However, there was evidence that even among these young (18- to 24-year-old) inductees, respiratory symptoms were more prevalent among persons from more polluted communities. Black and white inductees showed similar effects of pollution on bronchitis rates (Table 7.1.1). In the case of blacks, these effects were superimposed on higher base rates among persons residing in relatively clean outstate areas. Whether these high rates are attributable to sources of indoor pollution or other environmental factors, and whether these baseline rates are indeed verifiable, remains to be studied. Strangely, cigarette smoking alone was associated with no excess bronchitis in blacks, while cigarette smoking and air pollution combined accounted for more bronchitis than the additive effect of both pollution and smoking. Sample sizes among blacks in these categories were small. Among whites, air pollution and cigarette smoking were generally additive in their effect on bronchitis rates among smokers within polluted communities of each of the four CHESS areas.

Attempts were made to assess the length of residence in polluted areas required for development of excess bronchitis rates.[2-4] These findings should be accepted in a very preliminary vein because

### 7.1.3. EXCESS CHRONIC BRONCHITIS ATTRIBUTABLE TO AIR POLLUTION AND SMOKING, AND RELATIVE CONTRIBUTION OF EACH FACTOR

| Factor | Excess chronic bronchitis prevalence, percent[a] | | | |
|---|---|---|---|---|
| | New York | Salt Lake | Rocky Mountain | Chicago[b] |
| **Males** | | | | |
| Air pollution alone | 11.3 | 3.8 | 2.2 | 1.2 |
| Smoking alone | 9.3 | 16.6 | 15.8 | 13.4 |
| Air pollution + smoking | 17.1 | 23.8 | 17.4 | < 13.6 |
| Air pollution alone: smoking alone | 1:0.82 | 1:4.4 | 1:7.2 | 1:11.2 |
| **Females** | | | | |
| Air pollution alone | 4.0 | 2.2 | 1.5 | |
| Smoking alone | 11.9 | 12.8 | 10.7 | |
| Air pollution + smoking | 16.1 | 19.2 | 11.8 | |
| Air pollution alone: smoking alone | 1:3.0 | 1:5.8 | 1:7.1 | |

[a] Excess prevalence: absolute excess above rate experienced by nonsmokers in the less polluted community of the same CHESS area.

[b] Analysis restricted to whites because of small black sample size.

relatively small sample sizes were available for analysis after populations were subdivided into smoking- and residence-duration-specific groups. The overall evidence suggests that immigrants into polluted areas reported excess chronic bronchitis after 2 to 7 years of exposure. Further evidence from the New York study[5] indicated that movement from polluted to clean communities could effect a substantial decline in bronchitis rates, while migration into a polluted community seems to result in high bronchitis rates like those of the long-time residents of more polluted neighborhoods. These conclusions should be taken as hypotheses for further testing, but they justify some optimism about current efforts to improve air quality. An important feature of the CHESS program is the plan to resurvey residents of the more polluted neighborhoods during and after achievement of desirable air quality.[1] These studies can provide considerably more firmness to the conclusions stated in this monograph.

Other covariates such as age, race, sex, socioeconomic status, and occupational exposure were controlled, insofar as possible, by the selection of study areas and by appropriate adjustments in statistical analyses. Covariates other than occupational exposure played a relatively minor role as determinants of bronchitis prevalence. Participants with known occupational exposure were analyzed separately, and in none of the above quantitative assessments concerning air pollution and cigarette smoking was the occupationally exposed group included. Occupational exposure to irritating dusts, fumes, and aerosols added to the effects of ambient air pollution and cigarette smoking in producing a higher prevalence of chronic bronchitis among exposed workers.[2,3]

In general, occupational exposures made a quantitative contribution somewhat larger than that of air pollution and one-half as large as cigarette smoking.

Table 7.1.4 lists current (i.e. during the year of the survey) and past exposures (within 10 years)[6-9] estimated for those communities in which excess bronchitis was observed in the four CHESS studies. The precise exposure or dose that should be associated with excess respiratory symptoms could not be determined because accurate measures of past exposures were not made and the duration of exposure required to produce excess respiratory disease is not known. Current exposures may be taken as a worst case estimate for the chronic bronchitis effect, and past exposures as a least case estimate. In the best judgment of the investigators, excess chronic bronchitis in the Salt Lake Basin could be reasonably attributed to sulfur dioxide levels of 92 to 95 μg/m$^3$ and/or suspended sulfate levels of 15 μg/m$^3$. This was the only CHESS area in which low concentrations of total suspended particulates occurred in the presence of elevated sulfur oxide pollution. Pollutant concentrations measured in 1971 were unlikely determinants of the excess bronchitis rates in the High exposure Salt Lake Basin community. In the other CHESS areas, combinations of particulate matter and sulfur oxide exposures occurred, and the investigators judged that the lowest pollutant concentrations that could reasonably be associated with excess chronic bronchitis were past exposures to 100 to 177 μg/m$^3$ sulfur dioxide, 80 to 118 μg/m$^3$ total suspended particulates, and 9 to 14 μg/m$^3$ suspended sulfates. The individual contribution of each pollutant could not be identified.

From these data, it appeared that excess bronchitis may be reasonably associated with community exposures to sulfur oxides alone, in the form of annual levels of 92 to 95 μg/m$^3$ sulfur dioxide and 15 μg/m$^3$ suspended sulfates. When higher levels of particulate matter are present, annual exposures to 100 μg/m$^3$ sulfur dioxide, 120 μg/m$^3$ total suspended particulate, and 14 μg/m$^3$ suspended sulfate are reasonably associated with excess bronchitis. None of the CHESS areas experienced elevated exposures to total suspended particulates without concomitant increases in sulfur oxide levels. Overall, these data support the existing National Primary Ambient Air Quality Standards of 80 μg/m$^3$ annual mean (arithmetic) for sulfur dioxide and 75 μg/m$^3$ annual mean (geometric) for total suspended particulates. A National Standard for suspended sulfates has not been established.

## Summary of Lower Respiratory Disease Studies of Children

In the lower respiratory disease studies conducted in the Salt Lake Basin and Rocky Mountain CHESS areas,[10,11] three findings were consistently observed. First, for all combinations of disease and numbers of

**Table 7.1.4. RANGE OF POLLUTANT EXPOSURES ASSOCIATED WITH EXCESS CHRONIC BRONCHITIS**

| CHESS area | Current exposures (annual average), μg/m$^3$ | | | Exposures within past 10 years (annual average)[a], μg/m$^3$ | | |
|---|---|---|---|---|---|---|
| | $SO_2$ (80)[b] | TSP (75)[b] | SS (no standard) | $SO_2$ (80)[b] | TSP (25)[b] | SS (no standard) |
| Salt Lake | 62 | 66 | 12.4 | 92-95 | 53-70 | 15.0-15.3 |
| Rocky Mountain | 177-374 | 65-102 | 7.2-11.3 | 177-374 | 62-179 | 6.9-19.9 |
| Chicago | 96-217 | 103-155 | 14.5 | 100-282 | 118-177 | 14.1-17.3 |
| New York | 49.9-62.9 | 63.1-104.0 | 13.2-14.3 | 144-404 | 80-173 | 9-24 |

[a] Estimated from emissions data and pollutant trends.

[b] National Primary Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about 85 μg/m$^3$.

illness episodes, no significant association between total lower respiratory disease and pollution was found for children whose parents had been residents of their communities for less than 3 years. Second, for single and repeated episodes of croup and repeated episodes of any lower respiratory disease, families of children who had lived 3 or more years in the High exposure communities reported more illness across all ages of children from 0 to 12 years than did their counterparts in the less polluted communities. Third, for single and repeated illness episodes and for all residence durations, there were no associations of pollution exposure with pneumonia or number of hospitalizations for total lower respiratory diseases. The only inconsistencies noted were that for children who had lived 3 or more years in their community, both single and repeated episodes of bronchitis and single episodes of any lower respiratory diseases were significantly associated with pollution exposure in the Salt Lake study, whereas these associations were not found in the Rocky Mountain study.

The effects of the age and socioeconomic covariates were very consistent in the two lower respiratory disease studies. In almost every instance, significantly higher illness rates occurred at younger ages, while there were very few significant associations with socioeconomic levels of the household. In the Salt Lake study, no significant differences in illness rates of males and females were observed. In the Rocky Mountain study, male and female children who had lived in their communities for less than 3 years had similar illness rates, with the exception of single episodes of croup. But, in every instance, male children who had been residents of their community for 3 or more years had higher illness rates than females. The reason for this inconsistent sex effect is unknown.

The increase in the rates of single or repeated episodes of lower respiratory disease, croup, and bronchitis attributable to high air pollution exposure can be determined from the data in Table 7.1.5. This table gives the illness rates over a 3-year reporting period for children who had been residents of their communities for 3 or more years. During the 3-year periods covered by the two studies, the mean annual sulfur dioxide concentrations in the High exposure communities were 92 $\mu g/m^3$ in the Salt Lake Basin study and as high as 177 $\mu g/m^3$ in the Rocky Mountain study. Hence, a worst case estimate of the annual sulfur dioxide concentration associated with increased lower respiratory disease is 92 $\mu g/m^3$, while the least case estimate is 177 $\mu g/m^3$. (The National Primary Standard for sulfur dioxide is 80 $\mu g/m^3$ annual arithmetic mean.) During the same periods, mean annual suspended sulfate concentrations in the High exposure communities were 15 $\mu g/m^3$ in the Salt Lake Basin study and as low as 7.2 $\mu g/m^3$ in the Rocky Mountain study. For suspended sulfates, a worst case estimate of the annual concentration associated with increased lower respiratory disease is 7.2 $\mu g/m^3$ while the least case estimate is 15 $\mu g/m^3$. (A National Standard for this pollutant has not been established.) Total suspended particulate levels in the Rocky Mountain communities ranged from 65 to 102 $\mu g/m^3$, representing the worst case and least case estimates for this pollutant, respectively. (The National Primary Standard for total suspended particulates is 75 $\mu g/m^3$ annual geometric mean.)

It is interesting to note that larger increases in total lower respiratory disease and two of its components were observed in the High pollution community of the Salt Lake Basin study than in the corresponding communities in the Rocky Mountain study. Also, the mean annual suspended sulfate concentration was higher in the High pollution community in the Salt Lake Basin study than in the Rocky Mountain study; the opposite was true for sulfur dioxide. This suggests that increases in lower respiratory disease frequency are probably associated with suspended sulfates rather than sulfur dioxide.

Several factors should be remembered when interpreting the results of the lower respiratory disease studies. First, the data were collected by asking the children's parents about illness frequency over a 3-year period. Hence, the recall ability of the parents could affect the validity of the data, as could the degree of cooperation of the parents. However, it does not seem likely that this source of error affected the communities differently and thereby affected the community comparisons. Second, there could be differences in diagnostic criteria among the communities in a study. In both lower respiratory disease studies, a sample of physicians were asked to diagnose six respiratory syndromes so that a determination of differences in diagnostic criteria could be made. No differences were found in either study. Third, the communities observed in the studies were mainly white and middle class. Therefore, the results of these studies may not apply to other ethnic or socioeconomic groups. Fourth, a majority of the pollution exposure data in both studies were estimated from emissions data. The degree to which these give reasonable estimates of individual exposures may be questionable.

Table 7.1.5. AGE-, SEX-, AND SOCIOECONOMIC-STATUS-ADJUSTED 3-YEAR LOWER RESPIRATORY DISEASE ATTACK RATES PER 100 CHILDREN BY COMMUNITY, NUMBER OF EPISODES, AND STUDY AREA

| Disease category | Community | Number of episodes | Study area[a] | | | |
|---|---|---|---|---|---|---|
| | | | Rocky Mountain | | Salt Lake Basin[b] | |
| All lower respiratory disease | Low<br>High | ≥ 1 | 21.3<br>24.2 | (NS) | 27.6<br>38.2 | $(p < 0.001)$ |
| | Low<br>High | ≥ 2 | 9.2<br>12.1 | $(p < 0.001)$ | 15.7<br>23.4 | $(p < 0.001)$ |
| Croup | Low<br>High | ≥ 1 | 6.2<br>11.2 | $(p < 0.001)$ | 16.0<br>26.4 | $(p < 0.001)$ |
| | Low<br>High | ≥ 2 | 3.1<br>4.8 | $(p < 0.05)$ | 7.2<br>13.8 | $(p < 0.001)$ |
| Bronchitis | Low<br>High | ≥ 1 | 14.9<br>15.8 | (NS) | 16.8<br>23.6 | $(p < 0.001)$ |
| | Low<br>High | ≥ 2 | 5.1<br>6.7 | (NS) | 6.9<br>10.8 | $(p < 0.001)$ |

[a] Probability that the difference between the two rates is as large or larger than that observed under the hypothesis of no difference is given in parentheses. NS – not significant, $p > 0.05$.

[b] Rates given for low pollution exposure are weighted averages of the age-, sex-, and socioeconomic-status-adjusted rates for the Low, Intermediate I, and Intermediate II communities.

On the basis of the two lower respiratory disease studies summarized, and in the best judgment of the investigators, it seems reasonable to conclude that there is a positive association between lower respiratory disease frequency in children and pollution exposure, and that excess respiratory disease may reasonably be associated with community exposures to approximately 95 $\mu g/m^3$ sulfur dioxide and 15 $\mu g/m^3$ suspended sulfates. From these studies, there is no evidence that elevated levels of total particulate matter are required to produce the adverse effect.

## Summary of Acute Respiratory Disease Studies of Families

Table 7.1.6 summarizes findings for total acute respiratory disease (combined upper and lower tract disease) among family members in the Chicago and New York studies.[12, 13] With the exception of fathers, who often have greater occupational exposures and daily changes of exposure due to place of work, a consistent excess acute respiratory disease rate was reported among family members living in more polluted neighborhoods. The relative excess in acute respiratory illness rates within more polluted neighborhoods varied from 3 to 40 percent. A range estimate of 5 to 20 percent relative excess includes all but the most extreme values. Unfortunately, the Intermediate community for the Chicago study also had elevated pollutant concentrations of sulfur oxides and particulates; this community, therefore, does not afford a satisfactory baseline illness rate. In New York, the Intermediate I community consistently reported considerably higher illness rates in all family

### Table 7.1.6. RELATIVE RISK OF TOTAL ACUTE RESPIRATORY ILLNESS IN FAMILIES LIVING IN NEW YORK AND CHICAGO AREAS

| Family segment | Community | | Relative risk of total acute respiratory illness | |
|---|---|---|---|---|
| | Chicago | New York | Chicago | New York |
| Preschool children[a] | Intermediate | Low | 1.00 (9.37) | 1.00 (7.88) |
| | High | Intermediate I | 1.06 | 1.40 |
| | Highest | Intermediate II | 1.09 | 1.03 |
| School children | Intermediate | Low | 1.00 (4.56) | 1.00 (6.22) |
| | High | Intermediate I | 1.39 | 1.20 |
| | Highest | Intermediate II | 1.06 | 1.03 |
| Mothers | Intermediate | Low | 1.00 (5.00) | 1.00 (4.45) |
| | High | Intermediate I | 1.19 | 1.14 |
| | Highest | Intermediate II | 1.19 | 1.05 |
| Fathers | Intermediate | Low | 1.00 (3.09) | 1.00 (3.44) |
| | High | Intermediate I | 0.95 | 1.17 |
| | Highest | Intermediate II | 1.19 | 0.88 |

[a] For Chicago, data are for nursery school children only.

segments than did the Intermediate II community, even though measured pollutant concentrations were somewhat lower in the Intermediate I neighborhood. Other environmental factors, including the proximity of a large international airport, may have influenced the illness reporting of residents in the Intermediate I New York community. For these reasons, it was difficult to determine the magnitude of excess illness associated with specific pollutant levels in these two studies. A conservative estimate (i.e., closer to the least case than the worst case estimate) would be that exposures (Table 7.1.7) to 210 $\mu g/m^3$ sulfur dioxide with 104 $\mu g/m^3$ total suspended particulates and approximately 16 $\mu g/m^3$ suspended sulfates were associated with a 5 to 20 percent excess of acute respiratory illness in various family members. This estimate largely discounts the high illness experience of the Intermediate I New York community and the relatively low current sulfur dioxide levels in New York and Chicago. Further observations of acute respiratory illness in these and other CHESS areas are being made and should considerably refine the quantitative estimates given above.

The Chicago study[12] also provided evidence of increased susceptibility to epidemic $A_2$/Hong Kong influenza among otherwise healthy families exposed during the previous 3 years to atmospheric levels of 106 to 119 $\mu g/m^3$ sulfur dioxide, 151 to 159 $\mu g/m^3$ total suspended particulates, and 14 $\mu g/m^3$ suspended sulfates.

The effects of other factors on the incidence of acute respiratory disease (Table 7.1.8) were most interesting. In both Chicago and New York City areas, socioeconomic status was a significant factor in the incidence of upper respiratory disease; more illness was reported by respondents of the upper-middle socioeconomic level. No effect of socioeconomic level on reporting of lower respiratory disease was observed.

Although personal cigarette smoking was associated with a decrease in illness frequency in New York, it apparently had little effect on the initial contracting of respiratory infections, but smoking was a significant determinant in the development of lower tract illness as a result of the initial infection. Parental smoking also was a significant factor in the

Table 7.1.7. **RANGE OF POLLUTANT EXPOSURES IN NEW YORK AND CHICAGO COMMUNITIES HAVING EXCESS ACUTE RESPIRATORY DISEASE RATES IN FAMILIES**

| Period and pollutant | Annual average concentration, $\mu g/m^3$ | | | |
|---|---|---|---|---|
| | Chicago | | New York | |
| | High | Highest | Intermediate I | Intermediate II |
| Current exposures | | | | |
| $SO_2$ (80)[a] | 51 | 106 | 50 to 63 | 50 to 58 |
| TSP (75)[a] | 126 | 151 | 63 to 84 | 87 to 104 |
| SS (no standard)[a] | 14.5[b] | 14.5[b] | 13.2 | 14.3 |
| Previous 2 years | | | | |
| $SO_2$ | 83 | 119 | 144 | 210 |
| TSP | 135 | 159 | 80 | 104 |
| SS | 14.1[b] | 14.1[b] | 13.4 | 16.2 |
| Previous 5 years | | | | |
| $SO_2$ | 107 | 170 | 256 | 321 |
| TSP | 137 | 163 | 94 | 117 |
| SS | 14.4[b] | 14.4[b] | 23 | 23 |

[a] National Primary Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about 85 $\mu g/m^3$.

[b] Estimate from the Chicago stations of the National Air Sampling Network.

development of lower tract illness among nonsmoking young members of their households. This is an important association, providing additional evidence that smoking is more than a means of self-pollution and affects other individuals in the immediate environment as well.

Data collected during acute respiratory disease studies are difficult to interpret because, in addition to the effects of all other environmental factors, the incidence of illness depends first of all on exposure to infectious agents. The fact that many of these agents are more virulent than others in itself may affect the ease with which infections are recognized and reported. Also a mild attenuated agent may protect against infection with a more virulent one, thus the recognizable illness rate may depend upon the sequence in which successive exposures to infectious agents occur. These difficulties must be recognized, but controlling for them is impossible without a prohibitively large and expensive laboratory activity. Consequently, the credibility of the results depends not only on the careful collection and analysis of data, but also on the reproducibility of associations between increased illness and higher pollution exposure. This latter factor provides the greatest strength to the data reported in this monograph. The consistency with which increased illness rates were observed to be associated with higher pollution exposure levels in different parts of the country and in the various segments of the population add greatly to the credibility of the results.

Differences observed between metropolitan areas, e.g., between New York and Chicago, were anticipated to be greater than those that would be found between neighborhoods within a single area. These differences can be accounted for by the fact that data

### Table 7.1.8. SIGNIFICANCE OF EFFECTS OF SELECTED FACTORS ON ACUTE RESPIRATORY DISEASE

| Factor | Statistical significance of factor[a] | | | |
|---|---|---|---|---|
| | Upper tract disease | | Lower tract disease | |
| | Chicago | New York | Chicago | New York |
| Air pollution | $< 0.05$ | NS | $< 0.05$ | $< 0.005$ |
| Socioeconomic status | $< 0.001$ | $< 0.005$ | NS | NS |
| Personal cigarette smoking | NS | $< 0.005$[b] | $< 0.10$ | $< 0.005$ |
| Parental smoking effect on children | $< 0.10$ | $< 0.005$[b] | $< 0.001$ | $< 0.001$ |

[a] NS – not significant, $p > 0.10$.

[b] Associated with decrease in illness frequency.

were collected in each metropolitan area by different survey groups; thus techniques were consistent within the same area but may have varied somewhat from area to area as a result of differences in execution of the same study protocol. Furthermore, a more conservative definition of lower respiratory symptoms was employed in the Chicago than in the New York surveys. As a result, lower respiratory diseases were reported at lower frequencies in Chicago than in New York. These definitions have since been standardized for all CHESS areas.

## Summary of Pulmonary Function Studies

Ventilatory function of elementary school children, measured by the 0.75-second forced expiratory volume ($FEV_{0.75}$), was diminished in areas of elevated exposure to sulfur oxides. In all cases, observed decrements were subtle. In the New York study[14] only the older children (age 9 to 13 years) who had been exposed to substantially elevated pollutant concentrations for the first 5 to 10 years of life showed evidence of reduced ventilatory function. The best available estimates of these remote annual average exposures were as follows: sulfur dioxide, 131 to 435 $\mu g/m^3$; total suspended particulates, 75 to 200 $\mu g/m^3$; suspended sulfates, 18 to 28 $\mu g/m^3$.

From the New York study, the authors could not determine the relative importance of specific pollutants in reducing ventilatory function. From the Cincinnati study,[15] however, suspended sulfates emerged as a pollutant of particular concern. In all Cincinnati neighborhoods, sulfur dioxide concentrations were at or below the moderate level of 57 $\mu g/m^3$ during the time of testing, permitting suspended sulfates to be assessed in the relative absence of sulfur dioxide. Ventilatory function in white children exposed to suspended sulfate concentrations of about 9.5 $\mu g/m^3$ was lower than that of white children exposed to concentrations of about 8.3 $\mu g/m^3$. Black children in Cincinnati were all exposed to suspended sulfate concentrations of about 8.9 $\mu g/m^3$, and these children demonstrated no differences in ventilatory function.

On the basis of the Cincinnati study, it was conceivable that 1 year's exposure to 9 $\mu g/m^3$ of suspended sulfates, in the presence of moderate levels of sulfur dioxide and total suspended particulates (57 and 96 $\mu g/m^3$ respectively), might alone account for reduced ventilatory function. The New York study strongly indicated a more moderate interpretation, however. It was the authors' best judgment that 8 to 9 years of exposure to about 10 to 13 $\mu g/m^3$ of suspended sulfates might reduce ventilatory function. If these suspended sulfate exposures were accompanied by exposures to about 200 to 250 $\mu g/m^3$ of sulfur dioxide and about 100 to 150 $\mu g/m^3$ of total suspended particulates, further reductions in $FEV_{0.75}$ might be expected.

Clearly, these best judgment estimates are based on suggestive, nonconclusive evidence. In Cincinnati, for example, the socioeconomic-racial patterns of exposure to suspended nitrates were very similar to suspended sulfate exposure patterns. Though absolute levels of suspended nitrates were much lower than suspended sulfates, possible effects of suspended nitrates could not be ruled out. Also, the ventilatory performance of black children in Cincinnati remains somewhat confusing. At present, it is impossible to disentangle the effects of objective environmental factors from these children's possible subjective responses to the all-white testing teams.

The contribution of covariates to pulmonary function results in school children is summarized in Table 7.1.9. Height, age, sex, and race are well recognized in the literature as significant determinants of pulmonary function in children, and these variables were taken into account in analyzing the CHESS data. Table 7.1.10 summarizes the CHESS pulmonary function findings to date. These studies have been repeated in New York and other CHESS areas for 2 successive years and will be reported in subsequent papers.

## Summary of Threshold Estimates for Long-term Pollution Effects

Table 7.1.11 summarizes worst case, least case, and best judgment estimates of the pollution levels

Table 7.1.9. SUMMARY OF EFFECTS OF COVARIATES OBSERVED IN

CHESS PULMONARY FUNCTION STUDIES

| Covariate | Effect |
|---|---|
| Height | • In all studies, height was the most significant determinant of $FEV_{0.75}$, being somewhat less important in children aged 5 through 8 years (0.045 liter/inch) than in children aged 9 through 13 years (0.063 liter/inch). |
| Age | • In New York, children aged 9 through 13 years demonstrated area differences in $FEV_{0.75}$ while children aged 5 through 8 years did not.<br>• In all children tested, age was a significant determinant of $FEV_{0.75}$, being somewhat more important in children aged 5 through 8 years (0.045 liter/year) than in children aged 9 through 13 years (0.019 liter/year). |
| Sex | • In New York, area differences in $FEV_{0.75}$ were statistically significant for boys aged 9 through 13 years, but not for girls of the same age.<br>• The $FEV_{0.75}$ of boys was consistently higher than that of girls of the same age and height. |
| Race | • In Cincinnati, the $FEV_{0.75}$ of black children was consistently lower than that of white children.<br>• In Cincinnati, white children demonstrated area differences in $FEV_{0.75}$, while black children did not. |

**Table 7.1.10. SUMMARY OF FINDINGS IN CHESS PULMONARY FUNCTION STUDIES**

| Location | Time | Age group tested | Findings |
|---|---|---|---|
| Cincinnati | 1967-68 | Second grade | • White children exposed to average suspended sulfate levels of 9.5 $\mu g/m^3$ had lower $FEV_{0.75}$ than white children exposed to average suspended sulfate levels of 8.3 $\mu g/m^3$.<br>• The $FEV_{0.75}$ of black and white children was lowest in winter.<br>• The $FEV_{0.75}$ of black children did not vary with air pollution exposure.<br>• The $FEV_{0.75}$ of black children was consistently lower than that of whites. |
| New York | 1970-71 | All elementary grades | • The $FEV_{0.75}$ of white children aged 9 through 13 years, who had been exposed to high levels of sulfur oxides and particulates during the first decade of life, was lower than that of children who had not been so exposed.<br>• This finding was statistically significant in males, but not in females.<br>• The $FEV_{0.75}$ of children aged 5 through 8 years did not vary consistently with pollution exposure.<br>• The $FEV_{0.75}$ of children in all grades was lowest in winter. |

that can be associated with adverse effects on the health indicators discussed in the previous sections.

## RESPONSES TO SHORT-TERM POLLUTANT EXPOSURES

In contrast to the health indicators previously summarized in this paper, studies on panels of asthmatic and cardiopulmonary subjects gave the investigators the opportunity to relate daily changes in symptom status to daily changes in pollutant levels.

### Summary of Asthma Panel Studies

One pattern immediately emerged from the asthma studies conducted in the Salt Lake Basin and New York.[16,17] As shown in Table 7.1.12, daily asthma attack rates in the Salt Lake Basin were more consistently correlated with colder outdoor temperature than with any measured pollutant. Therefore, an analysis of asthma attack rates against daily pollutant concentrations was carried out within two ranges of minimum temperature, 30 to 50°F and greater than 50°F. These data are summarized for sulfur dioxide, total suspended particulates, and suspended sulfates in Figures 7.1.1 and 7.1.2 for Salt Lake and New York, respectively. Inspection of these figures reveals one quite consistent finding: asthma attack rates were most closely related to stepwise increases in the levels of suspended sulfates. Virtually no relationship between sulfur dioxide and attack rates appeared. Total suspended particulates (with the exception of $T_{min} >$ 50°F for New York) and suspended sulfates (with the exception of $T_{min}$ = 30 to 50°F for Salt Lake) were positively and stepwise correlated with daily asthma attack rates. In the Salt Lake Basin, where the effects of total particulates and suspended sulfates were partitioned, a higher frequency of asthma attacks was

### Table 7.1.11. SUMMARY OF CHESS STUDIES RELATING LONG-TERM POLLUTANT EXPOSURES TO ADVERSE EFFECTS ON HUMAN HEALTH

| Adverse effect | Type of estimate | Duration of exposure, years | Annual average levels linked to adverse health effects, $\mu g/m^3$ | | |
|---|---|---|---|---|---|
| | | | Sulfur dioxide (80)[a] | Total suspended particulates (75)[a] | Suspended sulfates (no standard)[a] |
| Increase in prevalence of chronic bronchitis in adults | Worst case | 3 | 62 | 65 | 12 |
| | Least case | 10 | 374 | 179 | 20 |
| | Best judgment | 6 | 95 | 100 | 15 |
| Increases in acute lower respiratory tract infections in children | Worst case | 3 | 92 | 65 | 7.2 |
| | Least case | 3 | 177 | 102 | 15 |
| | Best judgment | 3 | 95 | 102 | 15 |
| Increase in frequency or severity of acute respiratory illness in families | Worst case | 1 | 50 | 104 | 14 |
| | Least case | 3 | 210 | 159 | 16 |
| | Best judgment | 3 | 106 | 151 | 15 |
| Subtle decreases in childhood ventilatory function | Worst case | 1 | 57 | 96 | 9 |
| | Least case | 9 | 435 | 200 | 28 |
| | Best judgment | 8-9 | 200 | 100 | 13 |

[a] National Primary Ambient Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about 85 $\mu g/m^3$.

### Table 7.1.12. SIGNIFICANCE OF CORRELATION OF ASTHMA ATTACKS WITH ENVIRONMENTAL FACTORS

| Factor | Significance of correlation[a] | | | | | |
|---|---|---|---|---|---|---|
| | Salt Lake | | | New York | | |
| | Low | Intermediate | High | Low | Intermediate I | Intermediate II |
| Minimum temperature | $<0.01$[b] | $<0.01$[b] | $<0.01$[b] | NS | NS | $< 0.05$ |
| Total suspended particulates | NS | NS | $< 0.01$ | NS | NS | NS |
| Sulfur dioxide | NS | NS | NS | NS | NS | $< 0.01$[b] |
| Suspended sulfates | $< 0.05$[b] | NS | $< 0.01$ | $< 0.05$ | NS | NS |

[a] NS—not significant, $p > 0.05$.

[b] Inverse correlation.

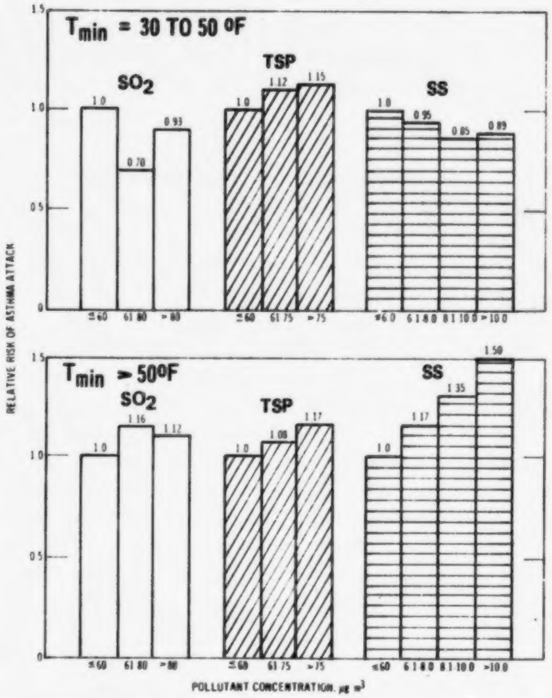

Figure 7.1.1. Relative risk of asthma attack versus sulfur dioxide, total suspended particulate, and suspended sulfate concentrations: Salt Lake area, two minimum temperature ranges.

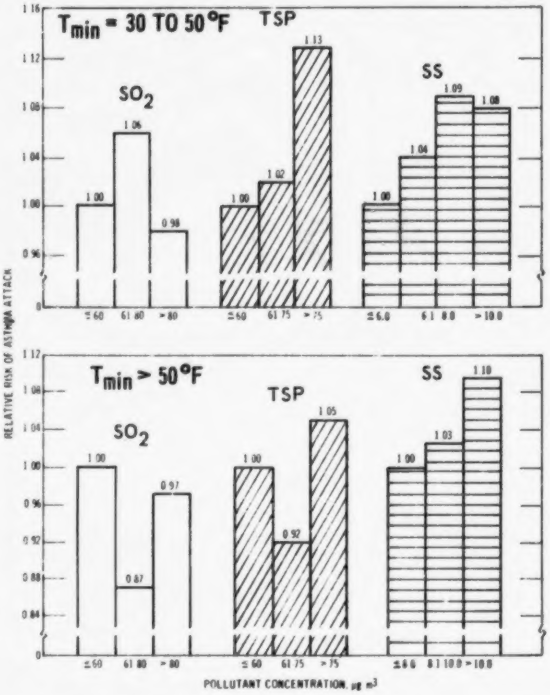

Figure 7.1.2. Relative risk of asthma attack versus sulfur dioxide, total suspended particulate, and suspended sulfate concentrations: New York area, two minimum temperature ranges.

observed at the same daily total suspended particulate concentration when a high sulfate fraction was present in the atmosphere (Figure 7.1.3). Thus, it appeared that sulfate levels were a stronger determinant of asthma attack rates than total suspended particulates were in the Salt Lake Basin. However, in the cold dry climate of the Basin, the effect of cold temperatures was considerably stronger than that of sulfates, and the pollutant threshold for the asthma response was much higher in colder than in more moderate temperatures (Figure 7.1.4).

In New York, asthma attack rates were more consistently associated with daily suspended sulfate levels than with either sulfur dioxide or total suspended particulates (Figure 7.1.2). As in Salt Lake, the pollutant threshold for the asthma response was higher in colder than in more moderate temperatures (Figure 7.1.5), but unlike Salt Lake, attack rates were generally higher on days with more moderate than with colder temperatures.

Thus, the effect of temperature was somewhat inconsistent between the two study areas; colder temperatures were associated with higher attack rates in the Salt Lake Basin but not in New York. It is difficult to compare the temperature effect in the two studies because they extended over different seasons of the year. In each case, the sulfate threshold was higher on colder days; and, in each case, elevated daily sulfate levels were quite consistently associated with increased asthma attack rates.

## Summary of Cardiopulmonary Panel Study

The pattern of daily aggravation of symptoms in cardiopulmonary subjects in New York[18] was very similar to that of asthma with respect to temperature and pollutants. In each of the three New York neighborhoods, cold temperatures were directly related to increased symptom rates in subjects with combined heart and lung disease (Table 7.1.13).

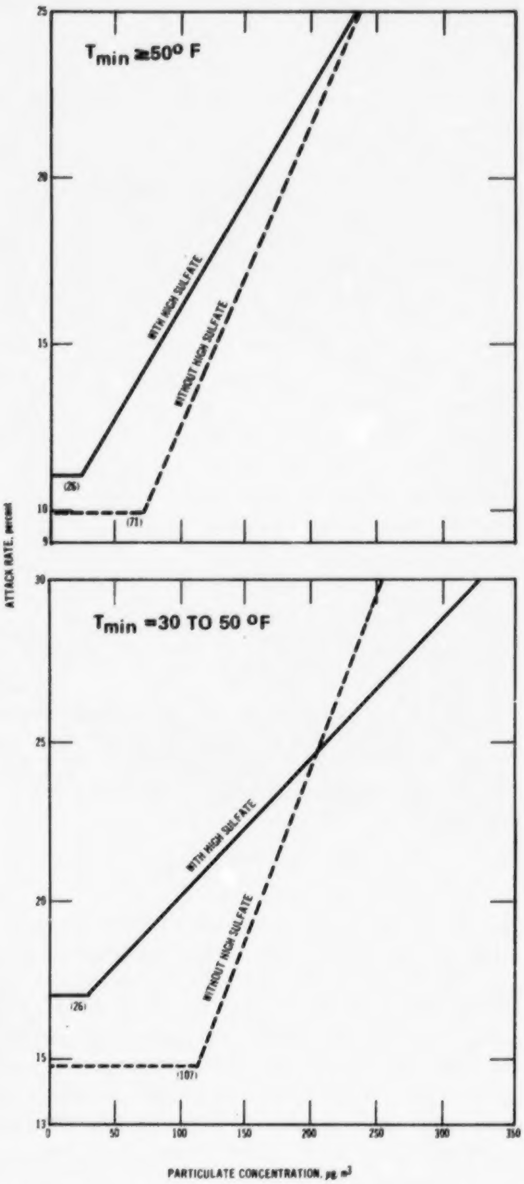

Figure 7.1.3. Effect of total suspended particulates with and without a high sulfate content on asthma attack rate: Salt Lake area, two minimum temperature ranges.

Elevated suspended sulfates were the only pollutant consistently associated with symptom aggravation, as shown in Table 7.1.13 and Figure 7.1.6. Daily sulfur dioxide and total suspended particulate concentrations could not be associated with symptom aggravation in the "heart and lung" panel, which was the most sensitive to variations in daily pollutant concentrations.

## Summary of Threshold Estimates for Short-term Pollution Effects

The pollutant thresholds for sulfur dioxide, total suspended particulate, and suspended sulfates among the several cardiopulmonary asthmatic panels at different temperature ranges are summarized in Table 7.1.14. Although this table presents pollutant thresholds for sulfur dioxide and total suspended particulates, the above discussion should make it clear that suspended sulfate levels demonstrated the only consistent relationship with daily aggravation of symptoms in these diseased panelists. Thus, while adverse effects were occuring at daily concentrations below the National Daily Primary Air Quality Standard for sulfur dioxide and total suspended particulates, the investigators would attribute these effects to suspended sulfate concentrations on those days rather than to those pollutants. It was the best judgment of the investigators that significant aggravation of cardiopulmonary symptoms could be attributed to 24-hour suspended sulfate levels as low as 8 to 10 $\mu g/m^3$ on both cooler days ($T_{min}$ = 20 to 40°F) and warmer days ($T_{min} >$ 40°F). The investigators intuitively felt that the chemical composition and particle size involved in sulfate exposures were critical determinants of the threshold for the adverse response. Since these sulfate-symptom relationships were manifested even in the less polluted communities of the Salt Lake and New York CHESS areas, there was evidence that suspended sulfates emanating from point or urban sources penetrated well beyond the suburban ring and adversely affected persons living in more distant communities. Such penetration might involve smaller respirable particles, acid mist, or other atmospheric transformation products of sulfur oxide emissions. The magnitude of such remote exposures and their overall health importance cannot yet be quantified.

## CONCLUSIONS

In this paper, we have summarized an original series of studies describing a variety of pollutant relationships with several health indicators. The pollutants of main concern were sulfur dioxide, total

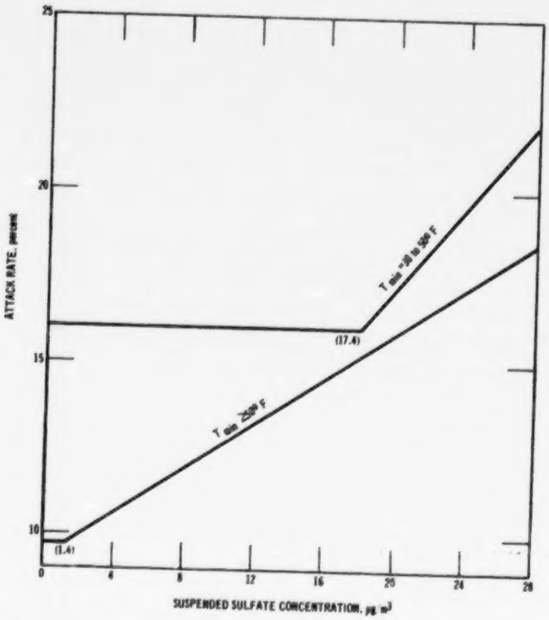

Figure 7.1.4. Effect of minimum daily temperature and suspended sulfates on daily asthma attack rates: Salt Lake area.

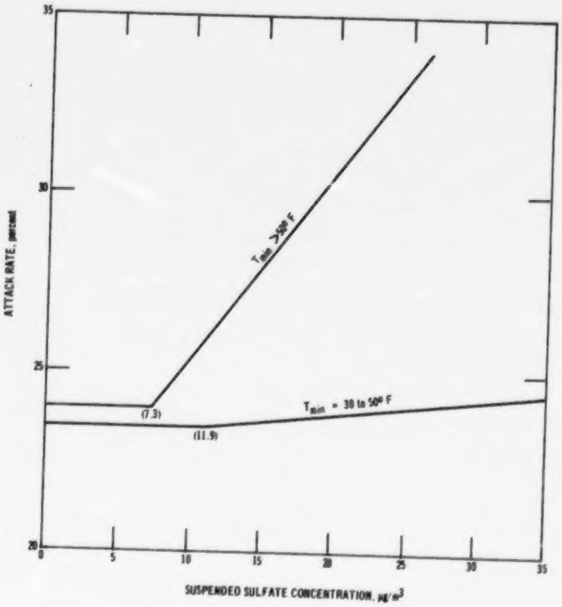

Figure 7.1.5. Effect of minimum daily temperature and suspended sulfates on daily asthma attack rate: New York area.

Table 7.1.13. SIGNIFICANCE OF CORRELATION OF CARDIOPULMONARY SYMPTOM AGGRAVATION WITH ENVIRONMENTAL FACTORS: NEW YORK AREA, "HEART AND LUNG" PANEL

| Factor | Significance of correlation[a] | | |
|---|---|---|---|
| | Low | Intermediate I | Intermediate II |
| Maximum temperature | < 0.01[b] | < 0.05[b] | < 0.01[b] |
| Total suspended particulates | < 0.05 | NS | NS |
| Sulfur dioxide | < 0.01 | NS | NS |
| Suspended sulfates | < 0.01 | < 0.01 | < 0.05 |

[a] NS—not significant, $p > 0.05$.

[b] Inverse correlation.

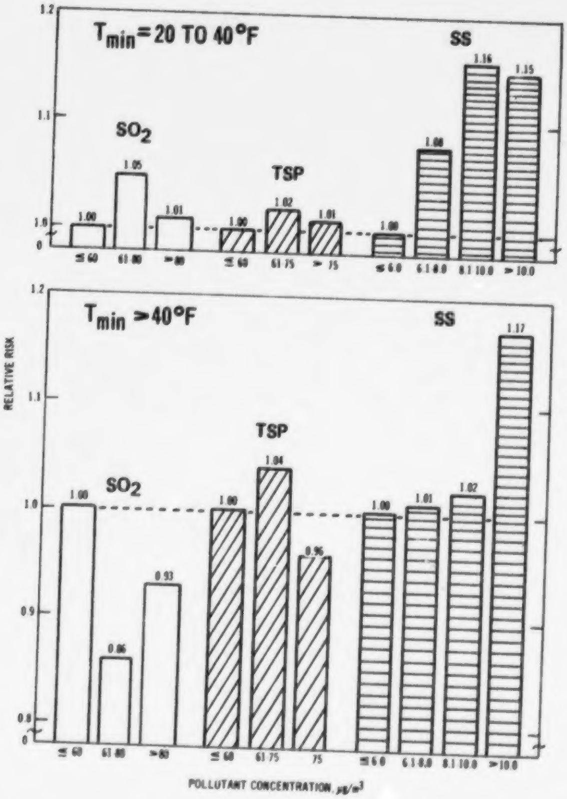

Figure 7.1.6. Relative risk of symptom aggravation versus sulfur dioxide, total suspended particulate, and suspended sulfate concentrations: New York area, two minimum temperature ranges, "heart and lung" panel.

suspended particulates, and suspended sulfates. We have examined the impact of community differences in exposure to these pollutants on chronic respiratory disease in adults, acute lower respiratory disease in children, acute respiratory illness in families, aggravation of symptoms in subjects with preexisting asthma and cardiopulmonary disease, and lung function of school children. These health indicators were selected because past studies by many investigators indicated that the frequency of these responses in a community was affected by sulfur oxides and particulates. Our studies more than substantiate these findings.

Our results can be divided into two groups: (1) health indicators responsive to cumulative long-term pollutant exposures and (2) health indicators sensitive to daily or shorter-term variations in pollutant exposure. Least case, worst case, and best judgment estimates concerning the pollutant thresholds for long-term exposures were given in Table 7.1.11. Best judgment estimates are recapitulated for long-term exposures in Table 7.1.15. Our findings support the existing National Primary Standards for long-term, or annual average, exposures, insofar as we have measured the desirability of these standards in terms of the health indicators mentioned.

With regard to short-term exposures, least case, worst case, and best judgment estimates were given in Table 7.1.14, and best judgment estimates are recapitulated in Table 7.1.16. Our data indicate that adverse effects on elderly subjects with heart and lung disease and on panels of asthmatics are being experienced even on days below the National Primary Standard for 24-hour levels of sulfur dioxide and total suspended particulates. However, as is evident from the presentation, these adverse health effects should be attributed to suspended sulfate levels rather than to the observed concentrations of those pollutants. The consistency of the relationship between symptom aggravation and sulfate levels, and the lack of consistency for this relationship with other pollutants, leads us to this conclusion.

Having identified atmospheric suspended sulfates as an environmental pollutant of present concern to health, we by no means have acquired sufficient intelligence to establish a National Standard for this pollutant. We know little about the environmental determinants of atmospheric suspended sulfates, and less about the means to control sulfate levels or to bring about significant reductions in sulfate concentrations in urban, suburban, and rural areas (particularly of the northeastern United States). In identifying the need for control of sulfates, we have raised a series of unstated questions and issues. Are all sulfates equally biologically reactive? Are sulfates reactive because of the chemical properties associated with specific chemical compounds, or because of physical properties such as particle size or pH? Are sulfates equally reactive in humid and dry air, and at warm and cold temperatures? These biological issues must be addressed and satisfactorily resolved, because our strategies to control sulfate levels may be critically dependent on the nature of the sulfate-biologic response relationship. If acid mist is the problem, we may be able to neutralize the sulfur oxides emitted at the source, without more stringent reductions in sulfur oxide emissions than are presently required to achieve National Primary Standards. On the other hand, if atmospheric transformation products of sulfur dioxide are implicated, we may be forced to restrict even more severely the sulfur

## Table 7.1.14. SUMMARY OF THRESHOLD ESTIMATES FOR ADVERSE EFFECTS OF SHORT-TERM EXPOSURES

| Adverse effect | Type of estimate | Minimum temperature, °F | Daily average levels linked to adverse health effects,[a] $\mu g/m^3$ | | |
|---|---|---|---|---|---|
| | | | Sulfur dioxide (365)[b] | Total suspended particulate (260)[b] | Suspended sulfate (no standard)[b] |
| Aggravation of cardiopulmonary symptoms in elderly | | | | | |
| "Well" panel | Worst case | 20-40 | 81-365 | NPE | < 1 |
| | Least case | | NE | NE | NE |
| | Best judgment | | NPE | NPE | 8-10 |
| | Worst case | > 40 | 81-365 | 68 | 2 |
| | Least case | | NE | NE | 10 |
| | Best judgment | | NPE | 80-100 | 8-10 |
| "Heart" panel | Worst case | > 40 | NPE | 76-260 | 10 |
| | Least case | | NE | NE | 10-20 |
| | Best judgment | | NPE | NPE | 10 |
| "Lung" panel | Worst case | 20-40 | NPE | 76-260 | 6 |
| | Least case | | NE | NE | NE |
| | Best judgment | | NPE | NPE | 10 |
| | Worst case | > 40 | NPE | 76-260 | 11 |
| | Least case | | NE | NE | 12 |
| | Best judgment | | NPE | NPE | 12 |
| "Heart and lung" panel | Worst case | 20-40 | 181 | 47 | 9 |
| | Least case | | NE | NE | 10 |
| | Best judgment | | NPE | 80-100 | 10 |
| | Worst case | > 40 | NPE | 76 | 6 |
| | Least case | | NE | NE | 17 |
| | Best judgment | | NPE | NPE | 10 |
| Aggravation of asthma | Worst case | 30-50 | NPE | 61-75 | 8 |
| | Least case | | NE | NE | NE |
| | Best judgment | | NPE | 105 | 9-10 |
| | Worst case | > 50 | 23 | 61-75 | < 1 |
| | Least case | | NE | NE | 10 |
| | Best judgment | | 180-250[c] | 70 | 8 |

[a] NE—no effect below Primary Standard, or simply no effect for suspended sulfates, for which no Primary Standard has been established. NPE—no proven effect below Primary Standard, or simply no proven effect for suspended sulfates.

[b] National Primary Air Quality Standard.

[c] This judgment is based on presently summarized studies and on a previously reported CHESS study of asthma in New Cumberland, West Virginia.[19]

## Table 7.1.15. BEST JUDGMENT ESTIMATES OF POLLUTANT THRESHOLDS FOR ADVERSE EFFECTS OF LONG-TERM EXPOSURES

| Effect | Threshold (annual average), μg/m³ | | |
|---|---|---|---|
| | Sulfur dioxide (80)[a] | Total suspended particulates (75)[a] | Suspended sulfates (no standard)[a] |
| Increased prevalence of chronic bronchitis in adults | 95 | 100 | 15 |
| Increased acute lower respiratory disease in children | 95 | 102 | 15 |
| Increased frequency of acute respiratory disease in families | 106 | 151 | 15 |
| Decreased lung function of children | 200 | 100 | 13 |

[a] National Primary Air Quality Standard. The particulate standard is a geometric mean; the equivalent arithmetic mean would be about 85 μg/m³.

## Table 7.1.16. BEST JUDGMENT ESTIMATES OF POLLUTANT THRESHOLDS FOR ADVERSE EFFECTS OF SHORT-TERM EXPOSURES

| Effect | Threshold, μg/m³ | | |
|---|---|---|---|
| | Sulfur dioxide (365)[a] | Total suspended particulate (260)[a] | Suspended sulfates (no standard)[a] |
| Aggravation of cardiopulmonary symptoms in elderly | >365 | 80-100 | 8-10 |
| Aggravation of asthma | 180-250 | 70 | 8-10 |

[a] National Primary Air Quality Standard.

content of fossil fuels. Until more definition of these issues is achieved, however, our findings strongly argue against any measures that would allow more sulfur loading of the atmosphere.

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# APPENDICES

# APPENDIX A:
# CHESS MEASUREMENT METHODS, PRECISION OF MEASUREMENTS, AND QUALITY CONTROL

This appendix presents the pollutant measurement methods employed by the Environmental Protection Agency's (EPA) Community Health and Environmental Surveillance System (CHESS), describes the quality control procedures employed to ensure optimum operation of the system, and presents results of duplicate testing to determine the precision of the measurements.

In recent years, additional types of pollutant measurement devices and automated or improved analysis methods have been incorporated in the CHESS system. In order to provide a complete and current description, these new methods and procedures, as well as those employed during the studies described in this report, are included.

## MEASUREMENT METHODS

### Sulfur Dioxide Determination (West-Gaeke Method)[1]

The method is the reference method as specified in the Federal Register.[2] CHESS sampling and analysis procedure deviated from the reference method only by using a flow rate of 500 ml/min in lieu of 200 ml/min, and 35 ml of absorbing solution in lieu of 50 ml.

*Hardware*

The 24-hour sulfur dioxide bubbler system is composed of relatively simple parts (Figure A.1). From an ambient air inlet, air is drawn through a 15-cm-long bubbler stem of 6-mm glass tubing. One end of the tubing is drawn to an inner tip diameter of 0.06 cm to control bubble size. The air then bubbles through 35 ml of a 0.04 *M* sodium tetrachloromercurate (TCM) solution contained in a 164- by 32-mm polypropylene tube. The airstream then passes through a glass wool moisture trap contained in a second polypropylene tube. The air next passes through a 23-gauge hypodermic needle, which behaves as a critical orifice controlling the flow rate at approximately 500 ml/min. A second moisture trap is placed between the needle and the vacuum source. Moisture traps are used to reduce corrosion for both the vacuum pump and needle. They also function as containers for overflow of TCM solution should the train be assembled incorrectly. A calibrated rotometer attached at the ambient air inlet is used to measure the flow rate at the beginning and end of a sampling period. A change in flow rate of ±50 ml/min voids a sample. If flow decreases below 400 ml/min, the system is adjusted.

*Principle and Applicability*

Sulfur dioxide is collected by bubbling air through a sodium tetrachloromercurate solution to form a solution of nonvolatile dichlorosulfitomercurate. The ion produced during sampling is determined colorimetrically by reacting the exposed absorbing reagent with formaldehyde and acid-bleached pararosaniline hydrochloride to form the intensely colored pararosaniline methyl sulfonic acid.

The method is applicable to collection of 24-hour samples in the field and subsequent analysis in the laboratory by manual or automated methods.

*Range, Sensitivity, Precision, Accuracy, and Stability*

The system obeys Beer's Law up to 1.0 absorbance units.

The error for the combined sampling and analytical techniques is ±10 percent in the concentration range below 26 $\mu g/m^3$, with increasing accuracy with concentration in the range of 26 to 2600 $\mu g/m^3$.

After sampling, the concentration of the dichlorosulfitomercurate ion decreases at an average rate of

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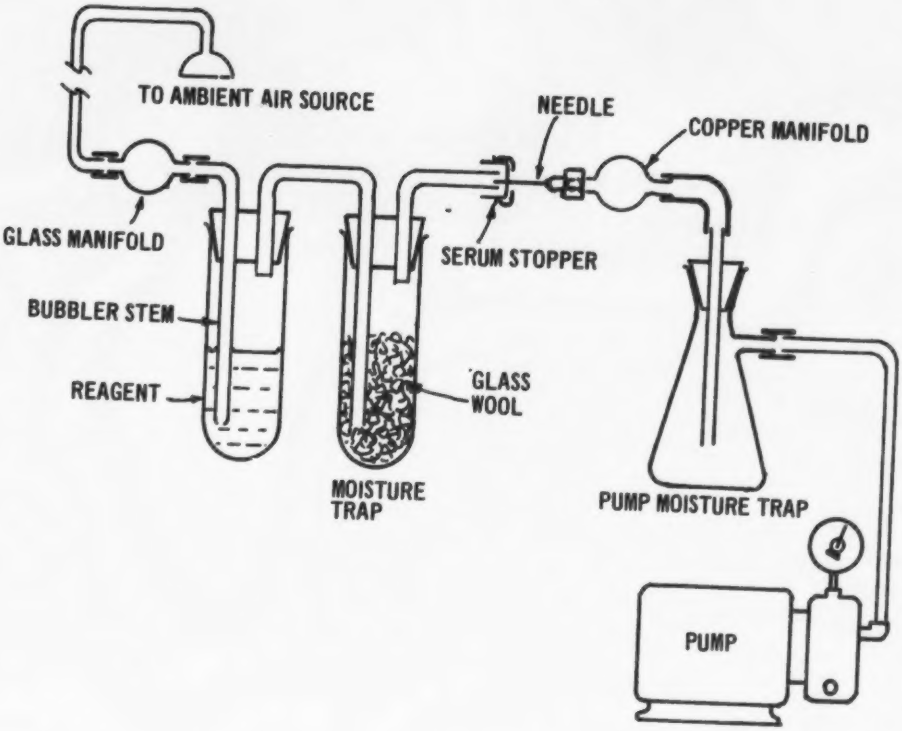


Figure A.1. Sulfur dioxide bubbler train.

1.5 percent per day. It is necessary, therefore, to analyze these samples with a minimum of delay.

*Analysis Apparatus*

The following equipment is required for the analysis:

1. Volumetric flasks – 100, 500, and 1000 ml.
2. Graduated cylinders – 100 ml.
3. Pipets – 1, 2, 3, 4, 5, 8, and 10 ml, volumetric transfer; 1 and 2 ml graduated in 0.1-ml intervals; 1 and 10 ml pipets are desirable but not necessary.
4. Test tubes.
5. Spectrophotometer or colorimeter capable of measuring absorbance or percent transmittance at 560 nm.

*Reagents*

*Sampling* – The absorbing reagent, 0.04 *M* sodium tetrachloromercurate (TCM), is prepared by dissolving 10.88 grams (0.04 mole) mercuric chloride and 4.68 grams (0.08 mole) sodium chloride in 1 liter of distilled water. (The solution is highly poisonous, and, if spilled, should be flushed from the skin with water immediately.)

*Analysis* – The following reagents are required for the analysis:

1. Sulfamic acid – 0.84 gram of sulfamic acid diluted to 100 ml with distilled water.
2. Formaldehyde – 0.5 ml of 38 percent formaldehyde diluted to 100 ml with distilled water. Solution is prepared weekly.

3. Stock pararosaniline hydrochloride (PRA) solution – 0.20 gram of PRA is dissolved in 100 ml of distilled water and filtered after 48 hours. The PRA used must have an assay of better than 95 percent and an absorbance maximum at 543 or 544 nm. The solution is stable for at least 3 months if stored in a dark, cool place.
4. Acid bleached PRA – The solution is prepared by pipetting 20 ml of stock PRA into a 100-ml volumetric flask, adding 6 ml of concentrated hydrochloric acid, and, after 5 minutes, diluting to 100 ml with distilled water. The color of the solution, which should be pale yellow with a greenish tint, is checked to assure that it has been mixed properly. In an amber bottle, the solution can be stored for a week at room temperature or for about 2 weeks under refrigeration.
5. Stock sodium thiosulfate solution (0.1 *N*) – The stock solution is prepared by dissolving 25 grams of sodium thiosulfate ($Na_2S_2O_3 \cdot 5H_2O$) in 1000 ml of freshly boiled, cooled, distilled water and adding 0.1 gram of sodium carbonate to the solution. The solution is allowed to stand for 1 day before standardization.
6. Sodium thiosulfate titrant (0.01 *N*) – 100 ml of stock sodium thiosulfate solution is diluted to 1000 ml with freshly boiled, cooled, distilled water. (Normality of resulting solution is equal to normality of stock solution times 0.100.)
7. Stock iodine solution (0.1 *N*) – The stock solution is prepared by mixing 12.7 grams of iodine, 40 grams of potassium iodide, and 25 ml of water in a 250 ml beaker. The solution is then transferred quantitatively to a volumetric flask and diluted to 100 ml with distilled water.
8. Iodine solution (0.01 *N*) – 50 ml of the stock iodine solution is diluted to 500 ml with distilled water.
9. Starch indicator solution – This solution is prepared by triturating 0.4 gram soluble starch and 0.002 gram mercuric iodide (preservative) with a little water and adding the paste slowly to 200 ml of boiling water. The solution is boiled until it is clear, cooled, and transferred to a glass-stoppered bottle.
10. Sulfite solution for standardization – 0.8 gram sodium metabisulfite ($Na_2S_2O_5$) is dissolved in 500 ml of recently boiled, cooled, distilled water. (Concentration is roughly 1000 μg $SO_2$/ml.)
11. Stock sulfite solution – By titration of the sulfite solution for standardization, as described in the following paragraphs on standardization, the amount of sodium metabisulfite needed to yield a solution with an accurate concentration of 1000 μg $SO_2$/ml is determined. A fresh solution of this amount of sodium metabisulfite in distilled water is prepared weekly.
12. Working sulfite standards – A solution of 10 μg $SO_2$/ml is formed by diluting 2.0 ml of the stock sulfite solution to 200 ml with 0.04 *M* TCM. By appropriate further dilutions with TCM, working standards in the range 0.1 to 1.0 μg/ml are obtained.

### *Standardization*

*Standard Sodium Thiosulfate* – This solution is prepared by standardization of the approximately 0.1 *N* solution of sodium thiosulfate (reagent 5 of the analysis section) prepared previously. First, 1.5 grams of primary standard potassium iodate (previously dried at 180 °C) is weighed to the nearest 0.1 mg and dissolved in water, and the solution is diluted to 500 ml in an iodine flask. Then 50 ml of this iodate solution, 2.00 grams of potassium iodide, and 10 ml of 1 *N* hydrochloric acid are mixed in a 500-ml volumetric flask. The flask is stoppered and allowed to stand for 5 minutes. The stock thiosulfate solution is then used to titrate the solution in the flask. The titration is continued until the solution becomes pale yellow. At this point, 5 ml of starch indicator (solution 9) is added, and the titration is continued until the blue color disappears. The normality of the sodium thiosulfate solution is calculated from the following equation:

$$N = 2.80\, W/V_t \qquad (A.1)$$

where

$N$ = normality of stock thiosulfate solution
$W$ = weight of potassium iodate, grams
$V_t$ = volume of thiosulfate required, ml

The factor 2.80 results from multiplying the conversion from grams to milligrams (1000) by the fraction of iodate used (0.1) and dividing by the equivalent weight of potassium iodate (35.67).

*Stock Sulfite Solution* – In determining the weight of sodium metabisulfite needed to produce a concentration of 1000 μg $SO_2$/ml for the stock sulfite solution (solution 10), two 500-ml iodine flasks, each containing 50 ml of 0.01 *N* iodine (solution 8) are used. To flask A (blank), 25 ml of distilled water is added; to flask B (sample), 25 ml of solution 10. The flasks are stoppered, allowed to react for 5 minutes, then titrated with 0.01 *N* sodium thiosulfate (solution 6). Titration is continued until each flask turns a pale yellow. Then 5 ml of starch solution 9 is added and the titration is continued until the blue color disappears. The concentration of sulfur dioxide in sulfite solution 10 is then calculated from the following equation:

$$C_1 = (32{,}000/25)(V_a - V_b)(N) \qquad (A.2)$$

where

$C_1$ = concentration of sulfur dioxide in solution 10, μg/ml
$V_a$ = volume of thiosulfate for blank, ml
$V_b$ = volume of thiosulfate for sample, ml
N = normality of thiosulfate (from Equation A.1)
32,000 = milliequivalent weight of sulfur dioxide, μg
25 = volume of sulfite solution, ml

The weight of sodium metabisulfite that will give the desired concentration of sulfur dioxide for the stock sulfite solution (solution 11) when dissolved in 500 ml of distilled water can then be calculated from

$$\frac{W_1}{W_2} = \frac{C_1}{C_2}$$

or

$$W_2 = W_1 (C_2/C_1) \qquad (A.3)$$

where

$W_2$ = weight of sodium metabisulfite needed for solution 11
$W_1$ = weight of sodium metabisulfite used for solution 10 (known)
$C_2$ = concentration desired for solution 11 (1000 μg/ml)
$C_1$ = concentration of solution 10 (from Equation A.2)

### *Analysis Procedure*

Samples are removed from the sampling train, properly sealed, and returned to the laboratory for analysis. At the laboratory, the sample is brought back to its original volume by the addition of distilled water to compensate for water loss during sampling. Then, 0.1 ml of sulfamic acid solution (solution 1) is added and mixed with the sample. In a test tube, 5 ml of the sample and 5 ml of TCM are mixed. Blanks consisting of 10 ml unexposed TCM are also prepared and treated in the same manner as the samples. Each sample is mixed with 1 ml of formaldehyde, then with 1 ml of bleached PRA. After 20 minutes to allow complete color development, the transmittance of the sample is determined at 560 nm on a colorimeter previously zeroed on the blank. At least three freshly prepared standards are run with each set of samples.

A manual method of analysis was used by CHESS until October 1972, after which analysis was accomplished on the Technicon Autoanalyzer. At the time of changeover, the TCM concentration was changed from 0.1 *M* to 0.14 *M*; the additional 0.04 *M* TCM was needed to prevent clogging in the Autoanalyzer. The change in TCM concentration was tested by EPA for accuracy and was also incorporated into the Federal Register method. Results of the TCM change caused no loss of precision in the analysis.

The sulfur dioxide concentration of the samples is determined from the following equation:

$$C_s = \frac{\log(\%T)(100)}{2.30258}(0.02tS_s + S_s) \qquad (A.4)$$

where

$C_s$ = sulfur dioxide concentration, μg/ml
%T = percent transmittance of sample from colorimeter
t = time between sampling and analysis, days
$S_s$ = slope of Beer's law curve determined from standards
2.30258 = conversion from natural to common logarithm
0.02 = average rate of decay of samples

A spectrophotometer, measuring absorbance rather than transmittance, can also be used. For absorbance, concentration is determined from the following equation:

$$C_s = A_s(0.02tS_s + S_s) \qquad (A.5)$$

where

$A_s$ = absorbance
$S_s$ = slope of Beer's law curve determined from standards
t = time between sampling and analysis, days
0.02 = average rate of decay of samples

## Nitrogen Dioxide Determination (Jacobs-Hochheiser Method)[3]

The method is the reference method as specified in the Federal Register.[4] CHESS sampling and analysis procedure deviated from the reference method only by using a flow rate of 500 ml/min in lieu of 200 ml/min and 35 ml of absorbing solution in lieu of 50 ml. In addition, two bubbler tubes in series with a restricted stem were used in lieu of one bubbler tube with a fitted stem.

### *Recent Evaluation of the Method*

Testing by EPA personnel in early 1972 revealed and identified apparent deficiencies with the Jacobs-Hochheiser method. Further reevaluation showed that the collection efficiency of the reference method varies nonlinearly with nitrogen dioxide concentration from 15 percent at 740 $\mu g/m^3$ to 50 to 70 percent at 20 to 50 $\mu g/m^3$. A second deficiency found is a positive interference caused by the presence of nitric oxide (NO) in the ambient atmosphere. Results of the evaluation, shown in Figure A.2 and Table A.1, have been summarized in the Federal Register[5] and an Environmental Science and Technology article.[6] The findings affect the range, sensitivity, interferences, and accuracy as specified in the reference method and noted in the following paragraphs. The concentration of nitrogen dioxide in samples collected from the ambient air, such as by CHESS monitoring stations, is in the range where it is questionable to assume a constant collection efficiency of 35 percent.

### *Hardware*

The 24-hour nitrogen dioxide gas bubbler system (Figure A.3) is very similar to the sulfur dioxide bubbler described previously. The air is bubbled through a sampling train of two 164- by 32-mm polypropylene tubes containing 0.1 *N* sodium hydroxide. Otherwise, components and operation are as described for the sulfur dioxide bubbler.

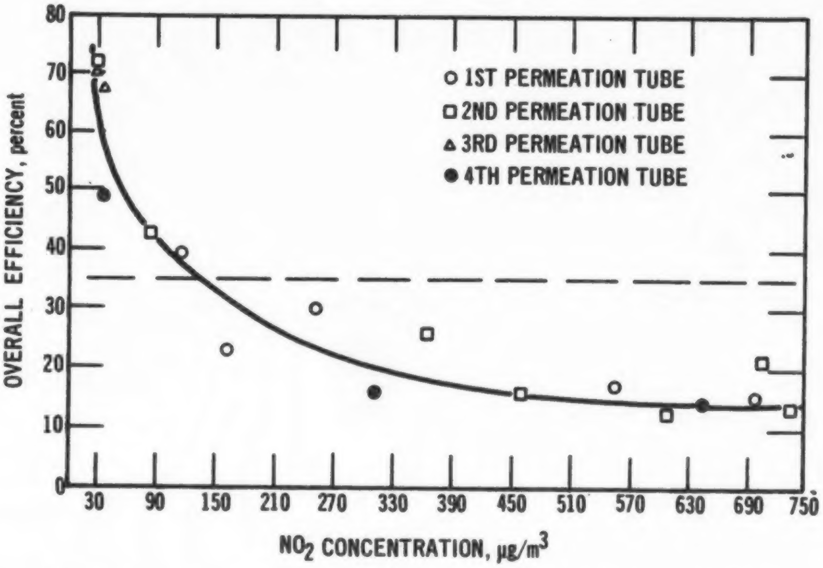


Figure A.2. Response to the nitrogen dioxide reference method during EPA evaluation.

Table A.1. EFFECT OF NITROGEN OXIDE ON THE REFERENCE METHOD FOR NITROGEN DIOXIDE

| Concentration, $\mu g/m^3$ | | Ratio $NO/NO_2$ | Expected $NO_2$ recovered, percent | Apparent $NO_2$ recovered, percent |
|---|---|---|---|---|
| $NO_2$ | NO | | | |
| 100 | 0 | 0.0 | 39 | 38 |
| 102 | 63 | 0.6 | 39 | 38 |
| 105 | 127 | 1.2 | 38 | 52 |
| 122 | 627 | 5.1 | 36 | 57 |
| 180 | 0 | 0.0 | 29 | 29 |
| 244 | 1205 | 4.9 | 24 | 45 |
| 248 | 1279 | 5.2 | 23 | 55 |
| 215 | 1242 | 5.8 | 26 | 50 |
| 311 | 0 | 0.1 | 20 | 17 |
| 316 | 111 | 0.4 | 20 | 30 |
| 318 | 332 | 1.1 | 20 | 33 |
| 356 | 1060 | 3.0 | 18 | 44 |

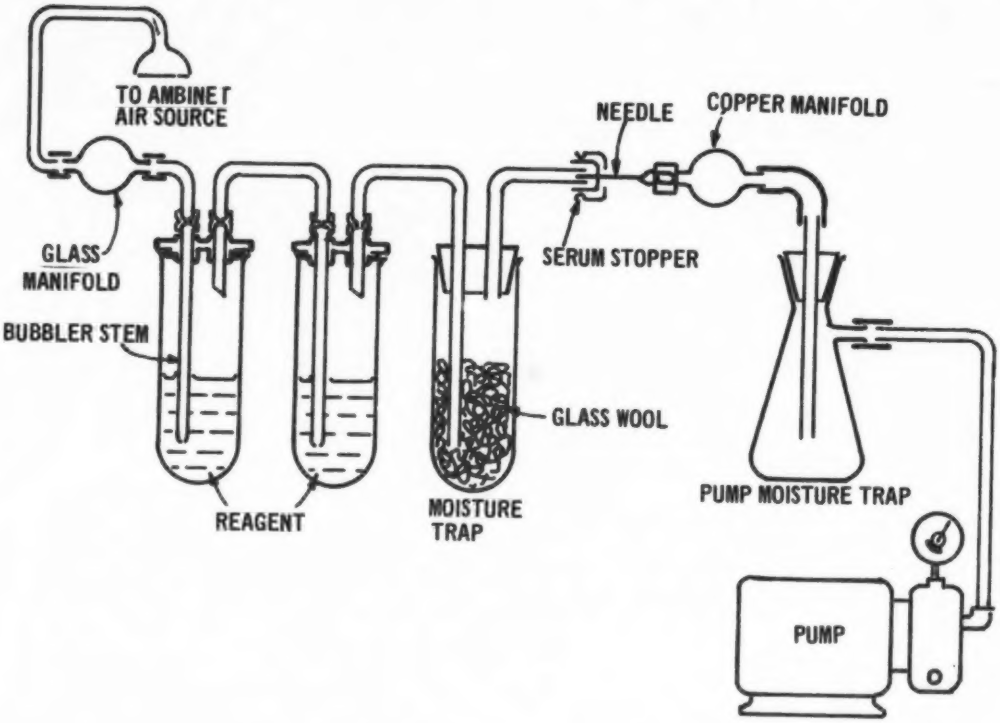


Figure A.3. Nitrogen dioxide bubbler train.

### *Principle and Applicability*

Nitrogen dioxide is collected by bubbling air through a sodium hydroxide solution to form a stable solution of sodium nitrite. The nitrite ion produced during sampling is determined colorimetrically by reacting the exposed absorbing reagent with phosphoric acid, sulfanilamide, and N-1-naphthylethylenediamine dihydrochloride (NEDA).

The method is applicable to collection of 24-hour samples in the field and subsequent analysis in the laboratory.

### *Range, Sensitivity, and Interferences*

The range of the analysis is 0.04 to 1.5 μg $NO_2$/ml. A concentration of 0.04 μg $NO_2$/ml will produce an absorbance of 0.02 using 1-cm cells. The interference of sulfur dioxide is eliminated by converting it to sulfuric acid with hydrogen peroxide before analysis.

### *Precision, Accuracy, and Stability*

The relative standard deviations are 14.4 and 21.5 percent, respectively, at nitrogen dioxide concentrations of 140 and 200 μg/$m^3$ based on an automated analysis of samples collected from a standard test atmosphere. The precision for manual analysis would probably be different, and no accuracy data are available for manual analysis. Samples are stable for at least 6 weeks.

### *Analysis Apparatus*

The following equipment is required for analysis:

1. Volumetric flasks – 50, 100, 200, 250, 500, and 1000 ml.
2. Graduated cylinder – 1000 ml.
3. Pipets – 1, 2, 5, 10, and 15 ml, volumetric; 2 ml graduated in 0.1-ml intervals.
4. Test tube.
5. Spectrophotometer or colorimeter – Capable of measuring absorbance at 540 nm; band width is not critical.

### *Reagents*

The absorbing reagent, 0.1 *N* sodium hydroxide, is prepared by dissolving 4.0 grams of sodium hydroxide in distilled water and diluting to 100 ml.

For the analysis, the following reagents are required:

1. Sulfanilamide – 20 grams of sulfanilamide is dissolved in 700 ml of distilled water, mixed with 50 ml of concentrated phosphoric acid (85 percent), and diluted to 1000 ml. This solution is stable for a month if refrigerated.
2. NEDA – 0.5 gram N-1-naphthylethylenediamine dihydrochloride is dissolved in 500 ml distilled water. This solution is stable for a month if refrigerated and protected from light.
3. Hydrogen peroxide – 0.2 ml of 30 percent hydrogen peroxide is diluted to 250 ml with distilled water. This solution can be used for a month if protected from light.
4. Standard nitrite solution – This solution is prepared by dissolving a calculated amount of desiccated sodium nitrite ($NaNO_2$, assay of 97 percent or greater) and diluting to 1000 ml with distilled water. The amount of sodium nitrite used to yield a solution with a nitrogen dioxide concentration of 1000 μg/ml is calculated by the following equation

$$G - 100(1.500/A) \qquad (A.6)$$

where

G = amount of sodium nitrite required, grams
A = assay, percent (thus requiring the 100 factor)
1.500 = gravimetric factor in converting $NO_2$ to $NaNO_2$

5. Working nitrite standards – A solution of 10 μg $NO_2$/ml is formed by dilution of 1 ml of the standard nitrite solution to 100 ml with 0.1 *N* sodium hydroxide. By appropriate further dilutions with sodium hydroxide, working standards of 0.05, 0.1, 0.2, 0.3, 0.4, and 0.5 μg/ml are obtained.

### *Analysis Procedure*

Sample tubes are removed from the sample train, properly sealed, and returned to the laboratory for analysis. At the laboratory, the sample is brought back to its original volume by the addition of distilled water to compensate for water loss during sampling. To 10 ml of the collected sample in a test tube, 1.0 ml of hydrogen peroxide solution, 10.0 ml of sulfanilamide solution, and 1.4 ml of NEDA solution are added, with thorough mixing after the addition of each reagent. A blank, using 10 ml of absorbing reagent, is prepared in the same manner. After a 10-minute color-development period, the absorbance of the sample is measured against the blank at 540 nm. At least three standards are run with each set of samples.

A manual method of analysis and the Technicon Autoanalyzer method were both used by CHESS until June 1, 1972, after which only the automated method was continued.

*Calculation*

The nitrogen dioxide concentration of the samples is determined from the following equation:

$$C_n = A_n S_n \qquad (A.7)$$

where

$C_n$ = nitrogen dioxide concentration, $\mu$g/ml
$A_n$ = absorbance of sample
$S_n$ = slope of Beer's law curve determined from standards

## Total Suspended Particulate Determination (High-volume Sampler)[7]

The method is the reference method as specified in the Federal Register.[8]

*Principle, Applicability, and Hardware*

The high-volume air sampler (Figure A.4) is used to collect relatively large quantities of particulate matter on filter papers. The airflow rate is sufficiently high to allow collection of a sample adequate for gravimetric and chemical analysis. The high-volume sampler has three basic parts: (1) the faceplate and gasket, (2) the filter adapter assembly, and (3) the air pump and motor unit.

The sampler pulls ambient air through a Gelman type A 20- by 25-cm (8- by 10-inch) glass fiber filter at the rate of 1130 to 1700 liters/min (40 to 60 ft$^3$/min). The motor operates on approximately 90-volt, 60-hertz alternating current utilizing a "buck or boost" transformer between the sampler and the power outlet.

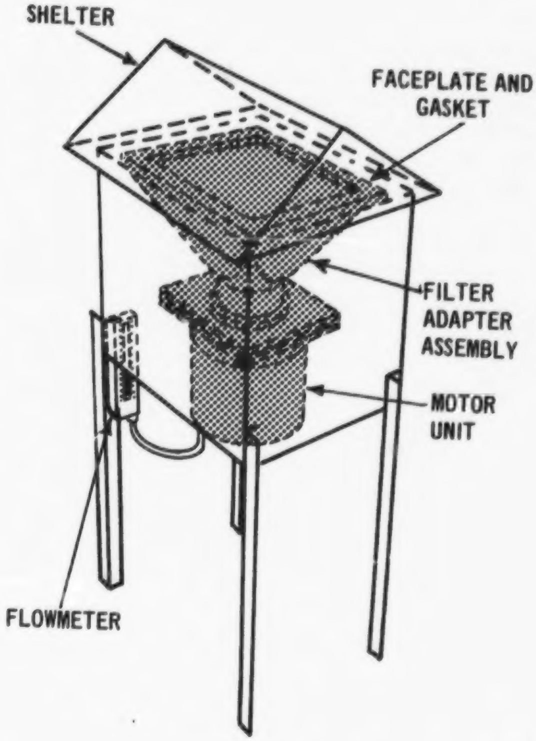


Figure A. 4. High-volume particulate sampler.

The sampler and rotameter are calibrated as a unit for accurate flow rate determinations. Start and stop flows are recorded for each sample. A change in flow rate greater than 40 liters/min voids the sample. Flow rates falling below 1130 liters/min are investigated and, depending on the cause, may void the sample.

Each unit is inspected and repaired every 25 calendar days in a program of preventive maintenance. A shelter of suitable design protects the sampler from adverse weather and vandalism.

*Range and Sensitivity*

When the sampler is operated at an average flow rate of 1700 liters/min for 24 hours, an adequate sample will be obtained even in an atmosphere having concentrations of suspended particulates as low as 1 $\mu$g/m$^3$. If particulate levels are unusually high, a satisfactory sample may be obtained in 6 to 8 hours or less. For determination of average concentrations of suspended particulates in ambient air, a standard sampling period of 24 hours is recommended.

Weights are determined to the nearest milligram, air flow rates are determined to the nearest 28 liters/min (1 $ft^3$/min), times are determined to the nearest 2 minutes, and mass concentrations are determined to the nearest microgram per cubic meter.

### *Interferences*

Particulate matter that is oily, such as photochemical smog or wood smoke, may block the filter and cause a rapid drop in airflow at a nonuniform rate. Dense fog or high humidity can cause the filter to become wet and severely reduce the airflow through the filter. Glass-fiber filters are comparatively insensitive to changes in relative humidity, but the collected particles can be hygroscopic.[9] If the water can be reversibly desorbed, there is no error in weighing because the filters are equilibrated at low humidity prior to weighing. Glass fiber filter media having a high pH can generate sulfate on the surface of the media if sulfur dioxide gas is present in the sampling atmosphere.

### *Precision, Accuracy, and Stability*

Based upon collaborative testing, the relative standard deviation (coefficient of variation) for a single analysis variation (repeatability of the method) is 3.0 percent. The corresponding value for multilaboratory variation (reproducibility of the method) is 3.7 percent.[9]

The accuracy with which the sampler measures the true average concentration depends on the amount of reduction of airflow rate and on the variation of the mass concentration of dust with time during the 24-hour sampling period.[8]

### *Analysis Apparatus*

The following equipment is required for analysis:

1. A balance room or desiccator maintained at 20 to 25 °C and less than 50 percent relative humidity for filter conditioning.
2. An analytical balance equipped with a weighing chamber designed to handle unfolded 20- by 25-cm filters and having a sensitivity of 0.1 mg.
3. A light source. Frequently a table of the type used to view X-ray film is used.
4. A numbering device capable of printing identification numbers on the filters.
5. Rootsmeter for airflow rate calibration.

### *Procedure*

Prior to sampling, each filter is exposed to the light source and inspected for pinholes, particles, or other imperfections. Particles are removed with a small brush, and filters with visible imperfections are rejected. The filters are equilibrated in the filter conditioning environment for 24 hours, and then weighed. Tare weight and filter identification number are recorded.

After sampling, the filter is again equilibrated for 24 hours, then reweighed. The weight of particulate matter collected is the weight of the filter after sampling minus its tare weight. After weighing, the filters may be subjected to detailed chemical analysis.

### *Calibration*

The instrument is routinely calibrated for accurate measurement of the volume of air sampled. A primary standard positive-displacement rotary meter is used in conjunction with a calibrating orifice-water manometer assembly. Details of calibration procedure are specified in the Federal Register.[8]

## Suspended Sulfate Determination

In the CHESS sampling program, the manual turbidimetric (Sulfaver) method of analysis for sulfate was used until September 1971. At that time, the methylthymol blue method replaced the Sulfaver method in order to automate the procedure, thus making possible a higher rate of analysis and reducing the chance for operator error.

### *Sample Collection and Extraction*

Suspended sulfate concentration is determined from ambient particulate matter collected by the standard high-volume sampler with glass fiber or other filter media. A sufficient quantity of particulate matter is collected in a typical 24-hour sampling period to allow chemical analysis for the sulfate ion. After the exposed filter is equilibrated and weighed, a 2- by 20-cm (3/4- by 8-inch) strip of it is placed in an erlenmeyer flask, 25 ml of distilled water is added, and the mixture is refluxed for 30 minutes. After the solution has cooled, it is filtered through acid-washed Whatman No. 42 (or equivalent) filter paper and then diluted to 50 ml with distilled water.

### *Turbidimetric Method*[10]

*Principle and Applicability*–An aqueous extract of the sample is treated with barium chloride in the presence of Sulfaver, a proprietary stabilizing agent, forming barium sulfate crystals of uniform size. The absorbance of the barium sulfate suspension is measured by a spectrophotometer or filter photometer. The sulfate ion concentration is determined by comparison to a previously determined standard calibration curve.

*Precision and Accuracy*–Based upon the analysis of 128 samples in triplicate, sulfate ion concentration determinations are reproducible within ±5.8 percent, producing a 95 percent confidence level, provided there is uniformly good technique and interferences are absent. The accuracy based upon recovery standards is estimated to be ±11.2 percent.

*Sensitivity*–Sensitivity may be increased by concentrating the sample or decreased by diluting it. The method is adequately sensitive for levels found in ambient environments.

*Interferences*–The accuracy of the method is changed when the concentration of the sample is changed since the size and shape of the barium sulfate crystals are affected by the strength of other ions present in the solution. In addition, if the concentration of sulfate is greater than 40 μg/ml of sample, the accuracy of the method decreases as the barium sulfate suspension loses stability. The standards recommended under standardization were selected to give maximum operating range. Reproducibility and accuracy are also affected by ion strength, pH, concentration of reagents, and temperature. For the indicated accuracy, the following conditions should be specified with respect to the conditions existing for preparation of the standard curve: (1) pH = ±1 unit, (2) temperature = ±5 °C, (3) Sulfaver added = 0.50 ± 0.01 gram, and (4) the specific conductance of aqueous extracts should not vary more than 300 μmhos/cm.

*Reagents*–All chemicals must be American Chemical Society analytical-reagent grade. The following reagents are required:

1. Sulfaver powder: A proprietary item (Cat. No. 396, Hach Chemical Co., Ames, Iowa), which contains barium chloride stabilizing and conditioning reagents.
2. Stock standard sulfate solution–A 500 μg $SO_4$/ml solution prepared by dissolving 0.74 gram anhydrous sodium sulfate ($Na_2SO_4$) in distilled water and diluting to 1000 ml in a volumetric flask.
3. Working standard sulfate solution–A 100 μg $SO_4$/ml solution prepared by diluting 10.0 ml of the stock standard to 100 ml in a volumetric flask with distilled water. This solution may not be stable for more than 24 hours; therefore, fresh solutions should be prepared immediately before use.

*Analysis Apparatus*–The following equipment is required for the analysis:

1. Refluxing apparatus–125-ml erlenmeyer flasks fitted with standard taper joints and matching water-cooled 200-ml-long condensers.
2. Heating apparatus–Electric hotplate with thermostatic control.
3. Filter paper–Whatman filter paper No. 42.
4. Photometer–Spectrophotometer or filter photometer for use at 500 nm with a light path of 1 to 5 cm for preparing standard sulfate solution.
5. Measuring spoon–0.5-gram capacity (Hach Chemical Co., Ames, Iowa).
6. Stirrer–Adams Cyclo-Mixer (or equivalent) with timer.
7. Graduated cylinders.
8. Volumetric flasks.
9. Pipettes.
10. Test tubes.
11. Cuvettes.
12. Spectrophotometer or colorimeter–Capable of reading absorbance in the 400-nm range for analyzing sample.

*Analytical Procedure*–From the filter extract, prepared as described earlier in this section, pipet 20 ml, or a suitable aliquot made up to 20 ml, into a 25- by 150-mm test tube. Add 1 level spoonful (0.5 gram) of Sulfaver and mix with the Cyclo-Mixer for exactly 60 seconds, making certain that the samples and standards are mixed the same length of time and at the same temperature. After at least 5 but less than 20 minutes, transfer the sample to a cuvette and read the transmittance. To correct for the filter and the reagents, subtract the absorbance of a distilled water blank carried through the same procedure.

Estimate the sulfate concentration in the 20-ml sample by referring the absorbance to the standard calibration curve, preparation of which is described in the next section.

*Standardization*–Prepare a series of standards from the working standard solution according to Table A.2.

**Table A.2. PROPORTIONS FOR MIXING STANDARDS FOR SUSPENDED SULFATE ANALYSIS**

| Working standard solution, ml | Distilled water, ml | Sulfate concentration, $\mu g$/20 ml |
|---|---|---|
| 0 | 20 | blank |
| 1 | 19 | 100 |
| 2 | 18 | 200 |
| 4 | 16 | 400 |
| 6 | 14 | 600 |
| 8 | 12 | 800 |

Construct, using rectangular coordinate graph paper, a calibration curve by plotting sulfate concentration (in $\mu g$/20 ml) against absorbance. The resulting plot should approximate a straight line. Stability of conditions should be checked by running a set of standard samples along with each batch of samples.

*Calculations*–The concentration of suspended sulfate is expressed as micrograms of sulfate per cubic meter of air at standard conditions (25 °C and 760 mm Hg). The following equation is used:

$$C_{ss} = A_r V_s C_l F/V$$

where

$C_{ss}$ = sulfate concentration, $\mu g/m^3$
$A_r$ = ratio of sample area to total exposed filter area (12)
$V_s$ = volume of filter extract (50 ml)
$C_l$ = concentration of sulfate in solution, $\mu g$/ml
F = dilution factor
V = volume of air sampled, $m^3$

or

$$C_{ss} = 600 C_l F/V \qquad (A.8)$$

### *Methylthymol Blue Method*[11]

*Principle and Applicability*–In this automated procedure for sulfate, the filter extract sample is first passed through a cation-exchange column to remove interferences. The sample containing sulfate is then reacted with barium chloride at a pH of 2.5 to 3.0 to form barium sulfate. Excess barium reacts with methylthymol blue to form a blue-colored chelate at a pH of 12.5 to 13.0. The uncomplexed methylthymol blue color is gray; if it is all chelated with barium, the color is blue. Initially, the barium chloride and methylthymol blue are equimolar and equivalent to the highest concentration of sulfate ion expected; thus the amount of uncomplexed methylthymol blue, measured at 460 nm, is equal to the sulfate present.[12]

*Range, Sensitivity, and Interferences*–The range of measurement is 0 to 50 $\mu g$/ml. The performance at 30 samples per hour using aqueous standards is as follows:

| | |
|---|---|
| Sensitivity (0.35 absorbance units) | 300 ppm |
| Coefficient of variation (95 percent confidence level at 200 ppm) | 1.4 percent |
| Detection limit | 10 ppm |

*Reagents*–The following reagents are required:

1. Barium chloride–Prepared by dissolving 1.5258 grams of barium chloride in 500 ml of distilled water and diluting to 1000 ml with distilled water. Solution is stored in a brown polyethylene bottle.
2. Methylthymol blue–Prepared by dissolving 0.1182 gram of methylthymol blue in 25 ml of barium chloride solution, then adding 4 ml of 1 *N* hydrogen chloride, which changes the color to bright orange. Finally, 71 ml of water is added and the solution is made up to 500 ml with ethanol. The pH of this solution is 2.6. It is stored in a brown glass bottle and prepared fresh daily.
3. Sulfate standard–Prepared by dissolving 1.4790 gram of sodium sulfate in 500 ml distilled water and diluting to 1000 ml with distilled water. For calibration, standards ranging from 10 to 50 $\mu g$/ml are prepared from this solution by serial dilutions.
4. Buffer (10.1 pH)–Prepared by dissolving 6.75 grams ammonium chloride in 500 ml of distilled water, adding 57 ml concentrated ammonium hydroxide, and diluting to 1000 ml with distilled water. After pH is adjusted to 10.1, solution is stored in a brown polyethylene bottle.
5. Buffered EDTA–Prepared by dissolving 40 grams of tetrasodium ethylenediamine tetraacetic acid (EDTA) in pH 10.1 buffer and diluting to 1000 ml with buffer. Solution should be stored in a brown polyethylene bottle.

6. Sodium hydroxide–Prepared by dissolving 7.2 grams of sodium hydroxide in 800 ml of distilled water, allowing solution to cool, and diluting it to 1000 ml with distilled water.

*Analytical Procedure*–Uncomplexed methylthymol blue is measured using a Technicon Autoanalyzer.[13] Since the measurements obtained do not conform to Beer's law, a Technicon Linearizer is also required to obtain readings that are directly proportional to concentration. Concentrations from the linearizer, in micrograms per milliliter, are converted to micrograms per cubic meter of air by Equation A.8.

Since cations, such as calcium, aluminum, and iron, would interfere with the measurement by complexing the methylthymol blue, these ions are removed by passage through an ion-exchange column. The column consists of a length of glass tubing 19 cm long, 2.0 mm inner diameter, and 3.6 mm outer diameter filled with the commercial ion-exchange resin Bio-Rex 70 (Bio-Rad Laboratories, New York, N.Y., 20-50 mesh, sodium form).

## Suspended Nitrate Determination

In the CHESS sampling program, the hydrazine sulfate-copper sulfate method of analysis for nitrate was used until December 1971. At that time, it was replaced with the copper-cadmium reduction method in order to refine the baseline stability of the instrumentation used. A Technicon Autoanalyzer was used for both methods.

### *Sample Collection and Extraction*

The suspended nitrate concentration is determined from ambient particulate matter collected on glass fiber filter media by the standard high-volume sampler. A sufficient quantity is collected in a typical 24-hour sampling period to allow chemical analysis for the nitrate ion. After the exposed filter is equilibrated and weighed, a 2- by 20-cm (3/4- by 8-inch) strip is cut and placed in an erlenmeyer flask, 25 ml of distilled water is added, and the mixture is refluxed for 30 minutes. After the solution has cooled, it is filtered through acid-washed Whatman No. 42 (or equivalent) filter paper, and then diluted to 50 ml with distilled water.

### *Hydrazine Sulfate-Copper Sulfate Method*[14]

*Principle and Applicability*–The nitrate ($NO_3$) is reduced to nitrite ($NO_2$) by hydrazine sulfate. Virtually 100 percent of the nitrate is reduced to nitrite after 10 to 15 minutes at 52 °C. The nitrite is converted to nitrous acid, which diazotizes sulfanilamide. The resulting complex is coupled with N-1-napthylethylenediamine dihydrochloride (NEDA) to produce an azo dye. The intensity of the dye color is measured at 540 nm on a colorimeter and the absorbance is related to concentration of suspended nitrate.

*Range and Sensitivity*–The range is 0.1 to 50.0 $\mu g$ $NO_3$/ml or approximately 0.03 to 15 $\mu g$ $NO_3/m^3$. The sensitivity is 0.1 $\mu g$ $NO_3$/ml or approximately 0.03 $\mu g$ $NO_3/m^3$ of air.

*Interferences*–The method is free from interferences normally encountered in procedures based on the nitration of aromatic compounds. Turbidity in the final solution will cause an erroneous increase in the apparent nitrate concentration. This effect may be eliminated by use of a spectrophotometer with a monochromatic light source.

Nitrites, if present, will react quantitatively and give false high nitrate values. Correction may be made by a parallel analysis for nitrite by substituting distilled water for the reducing agents, copper sulfate and hydrazine. Subtraction of the nitrite value from the total value gives nitrate concentration. Nitrite is usually a negligible interference.

*Precision, Accuracy, and Stability*–The standard deviation for the analytical portions of this method is ±0.02 $\mu g$ $NO_3$/ml for samples containing 1.0 $\mu g$ $NO_3$/ml. The color developed must be measured within 30 minutes after the addition of the diazotizing-coupling reagent.

*Analysis Apparatus*–The following equipment is required for the analysis:

1. Refluxing apparatus consisting of a 125-ml erlenmeyer flask fitted with standard taper joints and a matching water-cooled 200-mm-long condenser.
2. An electric hot plate with thermostatic control.
3. Graduated cylinders – 50 and 2000 ml.
4. Volumetric flasks – 50, 100, 500, and 1000 ml.
5. Pipettes – 1, 2, 5, 10, 20, 50, and 100 ml.
6. A spectrophotometer or colorimeter capable of reading absorbance at 460 nm. This may be part of an automated system.

*Reagents*–The following reagents are required for the procedure:

1. Sodium hydroxide (10.0 *N*) – Prepared by dissolving 400 grams sodium hydroxide in 500 ml freshly boiled and cooled (carbon dioxide-free) distilled water, allowing the mixture to cool, and diluting it to 1000 ml with carbon dioxide-free distilled water.
2. Sodium hydroxide (0.3 *N*) – Prepared by diluting 30 ml of the 10.0 *N* sodium hydroxide solution to 1000 ml with carbon dioxide-free distilled water.
3. Acetone solution (10%) – Prepared by diluting 100 ml acetone to 1000 ml with distilled water.
4. Diazotizing-coupling reagent – Prepared by completely dissolving 200 ml of 85 percent *O*-phosphoric acid and 80 grams sulfanilamide in approximately 500 ml distilled water, adding 4.0 grams NEDA and diluting to 2000 ml. Solution is stable for 4 to 5 weeks when stored in a dark bottle and kept in a cool place.
5. Stock copper sulfate solution – Prepared by dissolving 2.5 grams copper sulfate ($CuSO_4 \cdot 5H_2O$) in 500 ml distilled water and diluting to 1000 ml.
6. Working copper sulfate solution – Prepared by diluting 10 ml of the stock solution to 1000 ml with distilled water.
7. Stock hydrazine sulfate solution – Prepared by dissolving 13.73 grams hydrazine sulfate in water and diluting to 1000 ml. The solution is stable for 6 months or longer.
8. Working hydrazine sulfate solution – Prepared by diluting 50.0 ml of stock solution to 1000 ml with distilled water. The solution is stable for 1 month.
9. Standard nitrate solution – Prepared by dissolving 0.8153 gram anhydrous potassium nitrate in distilled water and diluting to 100 ml. This solution contains 50 μg $NO_3$/ml. After dilution, 1 ml chloroform is added for preservation. A fresh solution is prepared every week.

*Analysis Procedure*–For the manual method of analysis, 25 ml of the filter extract, prepared as described earlier in this section, is pipetted into a 50 ml volumetric flask. To this is added successively (since order of addition affects final color development), with complete mixing after each addition, 1 ml of working copper sulfate reagent, 1 ml of 0.3 *N* sodium hydroxide, and 1 ml of working hydrazine reagent. The flask is placed in a 52 °C water bath for 30 minutes (timed). The flask is removed and 2 ml of acetone solution and 6.0 ml of the diazotizing-coupling reagent are added with complete mixing after each addition. The solution is diluted to 50 ml with distilled water and mixed well. Absorbance is then measured against a true blank after at least 10 but not more than 30 minutes has elapsed.

This analysis may be automated. The sampling pattern for analysis is: sample, reagent-washout, sample; and the sampling rate is 45 samples per hour. Nitrate concentration in micrograms nitrate per milliliter is read from a standard curve prepared using standard solutions, described in the next paragraph, in the automatic analyzer.

*Calibration Standards*–By diluting 50.0 ml of the 500-μg $NO_3$/ml solution to 500 ml with distilled water, a solution containing 50 μg $NO_3$/ml is obtained. To obtain concentrations of 0.5, 1.0, 5.0, 10.0, 25.0, and 40.0 μg/ml, respectively, 1-, 2-, 10-, 20-, 50-, and 80-ml portions of the above solution are pipetted into a series of 100-ml volumetric flasks and diluted to the mark with distilled water. These standards are analyzed by either the manual or automated method. A plot of absorbance versus nitrate concentration gives a standard curve, which relates absorbance to concentration for ambient air samples.

*Calculation*–The concentration of suspended nitrate is expressed as micrograms of nitrate per cubic meter of air at standard conditions (25 °C and 760 mm Hg). The following equation is used:

$$C_{sn} = A_r V_s C_l F/V$$

where

$C_{sn}$ = nitrate concentration, μg/m$^3$
$A_r$ = ratio of sample area to total exposed filter area (12)
$V_s$ = volume of filter extract (50 ml)
$C_l$ = concentration of nitrate in solution, μg/ml
$F$ = dilution factor
$V$ = volume of air sampled, m$^3$

or

$$C_{sn} = 600 C_l F/V \qquad (A.9)$$

*Copper-Cadmium Reduction Method*[15]

*Principle and Applicability*–This automated procedure, performed with a Technicon Autoanalyzer, for the determination of nitrate and nitrite utilizes the procedure whereby nitrate is reduced to nitrite by a copper-cadmium reductor column.[15] The nitrite ion then reacts with sulfanilamide under acidic conditions to form a diazo compound. This compound then couples with N-1-napthylethylenediamine dihydrochloride (NEDA) to form a reddish-purple azo dye.

*Range, Sensitivity, and Interferences*–The concentrations of oxidizing or reducing agents and potentially interferring metal ions are well below the limits causing interferences. When present in sufficient concentration, metal ions may produce a positive error, i.e., divalent mercury and divalent copper may form colored complex ions having absorption bands in the region of color measurement. The range of measurement is 0 to 50 μg/ml.

The performance at 40 samples per hour using aqueous standards is as follows:

| | |
|---|---|
| Sensitivity (0.72 absorbance units) | 2.0 ppm |
| Detection limit | 0.04 ppm |
| Coefficient of variation (95% confidence level at 1.0 ppm) | 0.02% |

*Reagents*–Distilled water is not required for the preparation of all reagents for this analysis. However, if distilled water is not readily available, care is taken to ensure that the water used is completely free of contamination. Reagents are stored in glass bottles, and contact with air is avoided. The following reagents are required:

1. Ammonium chloride – First alkaline water is prepared by adding just enough ammonium hydroxide to distilled water to attain a pH of 8.5. Then 10 grams of ammonium chloride is dissolved in the alkaline water and diluted to 1000 ml. Finally, 0.5 ml of Brij-35 is added.
2. Color reagent – Prepared by completely dissolving (with heat if needed) 200 ml concentrated phosphoric acid and 20 grams of sulfanilamide in approximately 1500 ml distilled water. Then 1 gram of NEDA is dissolved in the solution and it is diluted to 2000 ml. Finally, 1.0 ml Brij-35 is added. Stored in a cold, dark place, the solution is stable for 1 month.
3. Stock standards – Prepared by dissolving 0.72 gram of potassium nitrate in distilled water and diluting to 1000 ml. The solution is stored in a glass bottle with a few drops of chloroform as a preservative. Working standards, ranging from 5 to 50 μg/ml are prepared from the stock standard daily by serial dilution.
4. Cadmium powder – Coarse cadmium powder, 99 percent pure. To remove grease and dirt, the fillings are rinsed with a little clean diethyl ether or 1 *N* hydrogen chloride followed by distilled water. The metal is then air-dried and stored in a well stoppered bottle.

*Reductor Column*–The reductor column is a U-shaped 35-cm (14-inch) length of 2.0-mm inside diameter glass tubing filled with cadmium powder that has been carefully washed with copper sulfate solution and distilled water to remove colloidal copper.

*Analysis*–Nitrate concentrations in micrograms nitrate per milliliter are obtained from a standard curve prepared using standard solutions in the automatic analyzer. Concentrations in micrograms per cubic meter of air are calculated by Equation A.9.

## Coefficient of Haze Determination (Optical Density of Filtered Deposit Method)[16]

### *Principle and Applicability*

The tape sampler (Figure A.5) is used to measure suspended particulate concentration of ambient air by the soiling index method. Air is drawn through a section of white filter paper, and the optical density of the resulting soiled spot is measured by comparison of the transmittance of the spot with that of a clean section of the same filter paper.

### *Sensitivity, Precision, and Accuracy*

The maximum amount of deposited matter that can be evaluated accurately by this method is that corresponding to 50 percent attenuation of incident light or a maximum optical density of about 0.30. For heavy particle loading, which will give rise to higher optical densities than the maximum range, the sampling cycle must be shortened accordingly. For lightly contaminated atmospheres, the sampling period may be lengthened.

Calibration of the gas flow meter, variations of the aspiration rate through the paper during sampling, the filtration efficiency of the collection media, and the accuracy of the optical density measurement must all be considered for accurate and precise measurements.

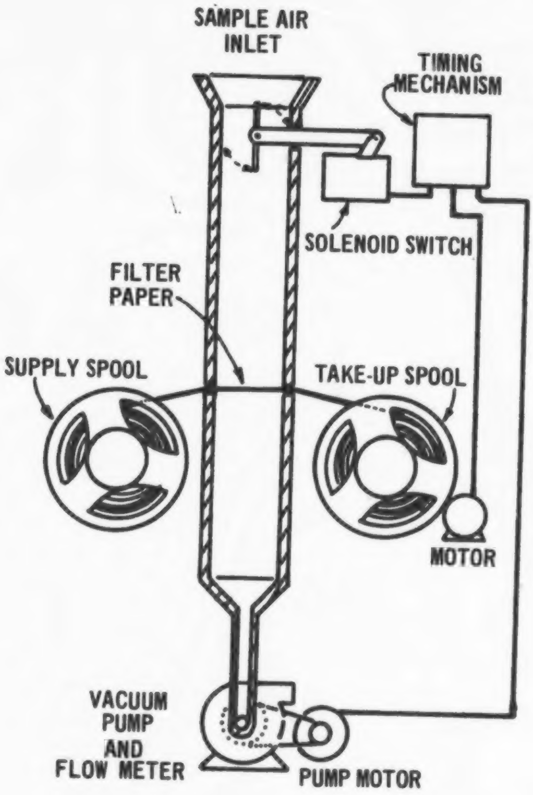


Figure A.5. Tape sampler.

*Hardware*

The tape sample contains the following functional subassemblies:

1. Sampling probe with filter screen.
2. Head with clamp to hold inlet tube to paper.
3. Paper holder with roll of filter paper.
4. Interval timer.
5. Calibrated flow meter.
6. Vacuum pump.
7. Spectrophotometer or photoelectric colorimeter for measuring optical density.

*Operation*

The air stream is passed through a 2.5-cm-(1-inch-) diameter area of the filter for a sampling period of 2 hours. The flow rate, nominally 7 liters/min (0.25 $ft^3/min$) is observed and recorded daily. At the end of the 2-hour sampling period, the filter paper is automatically advanced, exposing a clean area to the air stream for the next sample. A laboratory optical instrument such as the photoelectric colorimeter is used for determining the degree of soiling of the samples.

The flow meter of the tape sampler is periodically calibrated in the laboratory with a standard wet test gas meter. The calibration curve established is sent to the field with the instrument.

*Analysis*

The concentration of the sample is measured by optical density, expressed as log $(I_o/I)$, where $I_o$ is the intensity of light transmitted through a clean part of the filter paper next to the sample and I is the intensity of light transmitted through the sample. The computed optical density is converted to the coefficient of haze (COH) unit, which is 100 times the optical density. To account for the variables of flow rate and sample time, the size of the sample is expressed in linear units of air; specifically, the results are calculated as COHs per 1000 linear feet as follows:

$$\text{COHs/1000 lin ft} = 100\,[\log(I_o/I)] \div \frac{rt}{1000A_s} \qquad (A.10)$$

where

$I_o$ = intensity of light transmitted through clean filter paper
$I$ = intensity of light transmitted through sample
$r$ = flow rate, $ft^3/min$
$t$ = sample time, minutes
$A_s$ = area of filter spot, $ft^2$

Conversion:

1 lin ft = 30.5 cm
1 $ft^2$ = 930.25 $cm^2$
1 $ft^3$ = $2.84 \times 10^4$ $cm^3$

## Fine Particulate Determination (Cyclone Separator Method)[17,18]

*Hardware*

For separating the respirable fraction from the total suspended particle mass, a 1.25-cm-(0.5-inch-) diameter stainless steel cyclone collector is used as a prefilter to allow only the smaller respirable size particles to be collected on a filter.[18] The cyclone separator has been calibrated by several laboratories using aerosols of known size, density, electrostatic charge, size distribution, state of agglomeration, etc. The two most recent calibrations have been documented by the American Industrial Hygiene Association.[19,20]

For field sampling, the cyclone is connected in series with a downstream preweighed 27-mm-diameter filter and filter pad enclosed in an airtight plastic cassette (Figure A.6). Downstream of the cassette is a limiting orifice to control the flow rate at 9 liters/min (0.3 $ft^3$/min). A vacuum pump moves ambient air through the train.

In parallel with the cyclone sampler, an open-face 37-mm cassette simultaneously captures a total suspended particulate (TSP) sample on a similar 37-mm

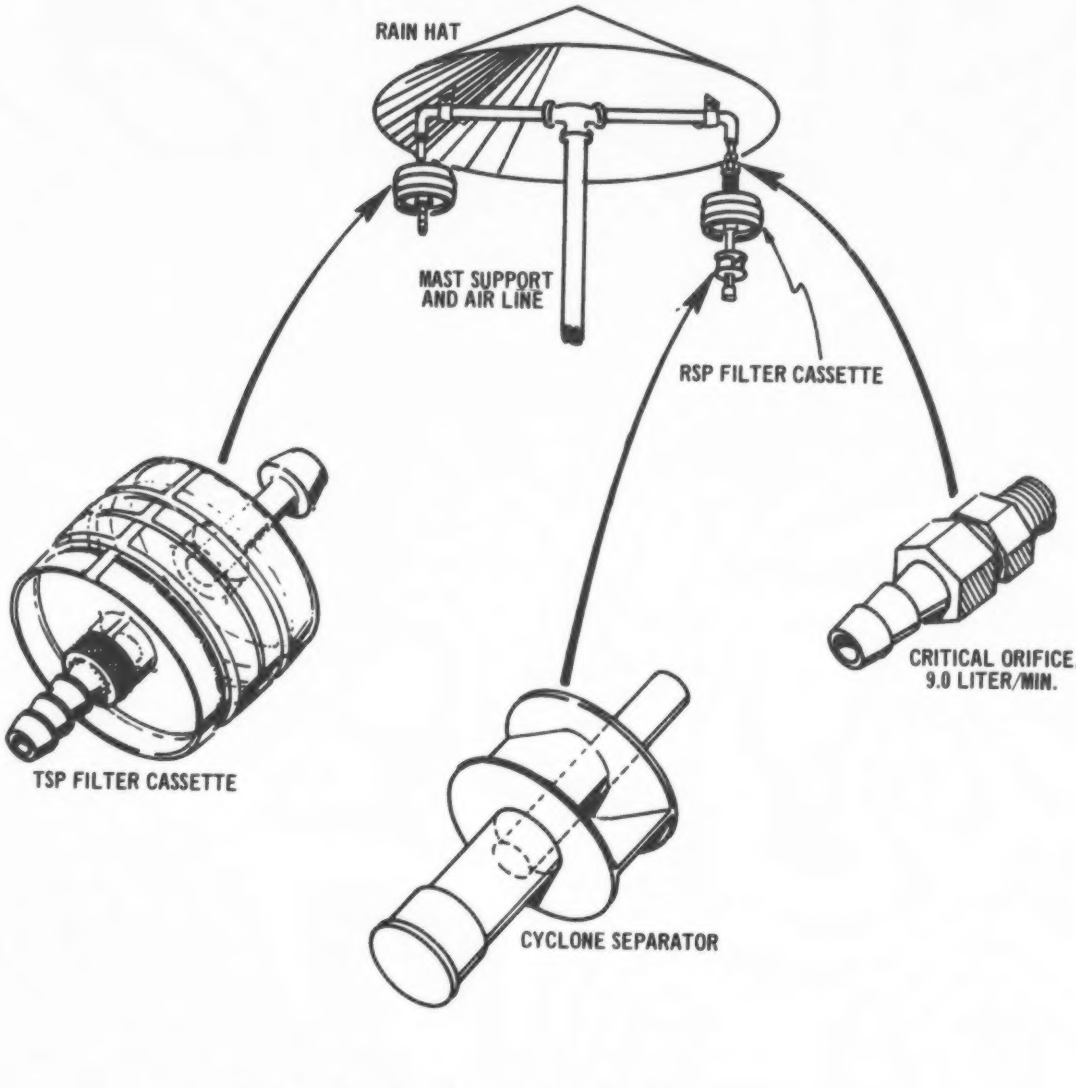


Figure A.6. Cyclone sampler and shelter assembly.

preweighed filter. The open-face cassette also has a limiting orifice of the same flow rate as the in-line cassette and cyclone so that comparative mass numbers are available for correlations. The total suspended particulate airflow is moved by the same vacuum pump that operates the cyclone sampler.

*Principle and Applicability*

Particles moving in an air stream tend to follow their original straight line direction of motion when the streamlines of airflow are deflected by an obstacle. Making use of this principle, a cyclone separator is utilized to separate the fine respirable suspended particulate (RSP) fraction of suspended airborne particles from the larger particles found in ambient air.[18] The air sample is drawn first through the cyclone where the larger particles are removed and discarded by impaction and settlement. The small particles follow the air vortex, pass through the top of the cyclone, and are captured on a filter for weighing and further analysis.

Mass concentration of both the fine fraction and total particulate matter found in ambient air is determined from the weight of samples collected and the volume of air passed through the train during each sampling period. Mass concentration is expressed as micrograms per cubic meter.

The fraction not passing through the cyclone separator represents the size particles that are filtered or trapped before reaching the uncilliated portions of the lungs. The portion of the sample collected on the cassette filter after the cyclone represents the particles that reach the lower regions of the respiratory tract. Shown in Figure A.7, along with collected particle sizes, is the fraction of each size particle not reaching the deep regions of the lungs. The curve was developed by the Atomic Energy Commission and has been accepted by other researchers, including the American Conference of Government Industrial Hygienist.[18]

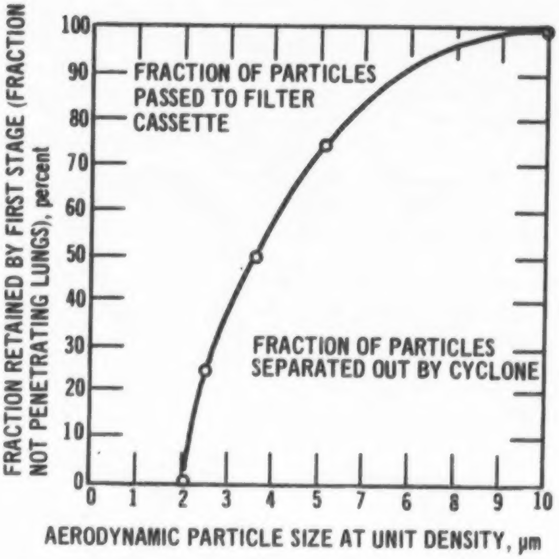


Figure A.7. Fractionation of fine particulates by cyclone separator.

Although the cyclone was designed primarily for use in industrial hygiene surveys, it has been adapted and proven to be successful for sampling mass concentrations of suspended particulates in ambient air. The critical orifice stabilizes air flow; therefore, a fairly representative sample is obtained with good repeatability.

*Range, Sensitivity, Precision, Accuracy, and Stability*

If the sampler is operated continuously for 24 hours at a flow rate of 9 liters/min, a meaningful sample will be obtained, provided the ambient air particulate concentration is at least 30 $\mu g/m^3$. Due to the small rate of flow, low-concentration sampling range is sensitive to weighing procedures.

Filter and sample weights are determined by using a precision balance. Weights are measured to the nearest 0.01 mg. Flow rates are measured to the nearest 0.1 liter/min. Real sampling times are recorded to the nearest minute.

High humidity or rainfall pulled into the filter may dissolve the portion of the sample that is water soluble.

The filter does not have water vapor gathering properties; however, the particles themselves can be hygroscopic and can introduce errors in the weight determinations if care is not exercised in equilibrating the samples to a fixed environmentally controlled humidity and temperature level before weighing.

Results of duplicate sampling at the Durham, North Carolina, Ambient Air Station has given a correlation coefficient of 0.97 for cyclone samplers operated simultaneously side by side, and a correlation coefficient of 0.98 for the open face TSP cassettes operated simultaneously side by side. At an average mass concentration of 115 $\mu g/m^3$ of particulate matter in ambient air, the standard deviation is 4.9 $\mu g/m^3$ (corresponding to a relative standard deviation of 4.3 percent); at an average of 39 $\mu g/m^3$ the standard deviation is 5.1 $\mu g/m^3$ (corresponding to a relative standard deviation of 13 percent); deviations apply to both RSP and TSP sampling.

The accuracy with which the samplers (RSP and TSP) measure the true average concentrations depends upon the degree of constant airflow rate maintained in the samplers and the accuracy in making weight determinations of collected samples. The vacuum pump used in sampling maintains enough pressure differential to keep the orifices critical at all times. Field sampling has shown that during 24-hour sampling periods flow rates vary less than 2.0 percent. This corresponds to the accuracy of the rotometer used in measuring the flow rates.

For both the RSP and TSP open-face cassettes, mass concentration measurements are very dependent on the accuracy of weight determinations of the filters. A series of repeated weight determinations for samples of the same magnitude as field samples (approximately 0.08 gram) has shown a standard deviation of $6.7 \times 10^{-5}$ grams. The accuracy increases very rapidly for concentrations above 60 $\mu g/m^3$.

### *Sampling Apparatus*

The following equipment is required for operation:

1. An open-face cassette and retaining ring with a 37-mm glass fiber filter and filter pad.
2. A cyclone separator backed by a 37-mm glass fiber filter and filter pad in a three-section cassette.
3. Two critical orifices for flow control.
4. One pump, with pressure gauge.
5. Flow and differential pressure measuring devices.

The vacuum pump must be capable of passing air at a rate of 9 liters/min through two clean 37-mm-diameter circular glass fiber filters connected in parallel. It must also maintain enough vacuum (approximately 50 cm Hg) to ensure that the two orifices will remain critical.

### *Analysis Apparatus*

For analysis, an environmentally controlled room, maintained at 25 °C and less than 40 percent relative humidity, and a balance with a sensitivity of 0.01 mg are required.

### *Reagents*

Filter media and filter-support material are needed. Glass fiber filters having a collection efficiency of 99.7 percent for particles of 0.3 $\mu m$ diameter are suitable for the quantitative measurement for concentrations of RSP and TSP. Other filter media, such as membrane filters, may be used also. Filter-support material must be a type of porous material suitable for filter support and flow distribution.

### *Sampling Procedures*

Prior to sampling, the filters are equilibrated to the conditions of the balance room for 24 hours and weighed to the nearest 0.01 mg. After sampling, the filters are again equilibrated for 24 hours and weighed to the nearest 0.01 mg. Sample weight is the weight obtained minus the tare weight of the filter.

### *Maintenance*

To ensure proper operation of the sampler, the following maintenance procedures are followed:

1. Vacuum pump. Inspect daily and keep ample supply of oil in well for lubrication of the pump. Replace vanes and lubricating accessories as needed.
2. Cyclone sampler. Clean daily with ethyl alcohol to ensure particle removal.
3. Rotometer. Inspect monthly. Clean, using alcohol, upon any indication that ball is sticking.
4. Critical orifices. Inspect daily. Clean with acetone weekly, or anytime flow drops below 9 liters/min.

### *Calibration Procedures*

The critical orifice flow rate of 9 liters/min must be verified before the orifices are installed in the field. The rotometer used to check the flow rate while the samplers are operating in the field must also be periodically calibrated.

*Calculations*

The true airflow rate is calculated from the equation:

$$Q = V/T \qquad (A.11)$$

where

$Q$ = airflow rate, $m^3/min$
$V$ = volume of air sampled, $m^3$
$T$ = sampling time, min

The volume of air sampled is determined from the equation:

$$V = \frac{Q_i + Q_f}{2} \times T \qquad (A.12)$$

where

$Q_i$ = initial airflow rate, $m^3/min$
$Q_f$ = final airflow rate, $m^3/min$

The mass concentration of suspended particulates (TSP or RSP) is determined by the following equation:

$$C_p = \frac{(W_f - W_i) \times 10^6}{V} \qquad (A.13)$$

where

$C_p$ = mass concentration of suspended particulate, $\mu g/m^3$
$W_i$ = initial weight of filter, grams
$W_f$ = final weight of filter, grams
$10^6$ = conversion of grams to micrograms

## Size Distribution Determination for Fine Particulates (High-volume Cascade Impactor Method)[17]

*Principle and Applicability*

An inertial impactor operates on the principle that, with enough momentum, particles moving in an air stream tend to follow their original straight line direction of motion when the air stream is deflected or curved in direction, and thus will be impacted on a surface in their path. Discrimination of particles by their momentum results in particle size classification since momentum is directly related to the particle size, shape, and density.

In the cascade impactor, a series of parallel plates containing a set number of sized holes are stacked with selected spaces between each plate. Also, between each pair of plates is a replaceable collection medium for collecting the impinged particles. The holes in each plate are offset from the holes in the succeeding plate in order to abruptly bend the air stream. In addition, the holes get smaller with each consecutive plate in order to increase the velocity step-wise with each plate, thus creating increasing levels of momentum for the particles left in the air stream.

*Hardware*

The impactor sampler (Figure A.8) is a multistage, multijet unit consisting of five multiorifice aluminum plates separated by sized neoprene rubber gaskets. At the end of the train of five plates is a standard 20- by 25-cm (8- by 10-inch) high-volume sampler filter to collect submicron particles. Each plate is approximately 30 cm (12 inches) in diameter and contains 300 uniform-sized jets. The alignment of the 30-cm plates and resulting airflow patterns direct the particles onto the surface of the next plate in series below. The collection surface is covered with a cut and perforated collection medium, such as glass fiber or aluminum foil, which has been preconditioned and weighed. The size and location of the jets are held to very close tolerances to give proper particle size fractionation. The collection medium contains punched holes so that it provides a collection surface for the plate above, but does not obstruct the jet openings in the plates on which it is mounted. The head interface plate contains four dowel pins and a center post for properly aligning the plates with respect to each other and with the collection medium. The assembled head is positioned on a 23- by 28-cm (9- by 11-inch) interface gasket and attached to a conventional high-volume filter holder inside the high-volume shelter. A standard high-volume air mover is used to provide airflow through the system. Regulation of the airflow through the size fractionator is achieved by adjusting a variable voltage transformer and noting the corresponding pressure drop through the assembled unit as indicated by a manometer. To collect particles accurately in predetermined size fractions, the fractionator should operate at 565 liters/min (20 $ft^3/min$). Since most of the particulate sample is collected on the upper stages of the sampler where airflow does not penetrate the collection medium, decreases in flow rate during sampling are minimized.

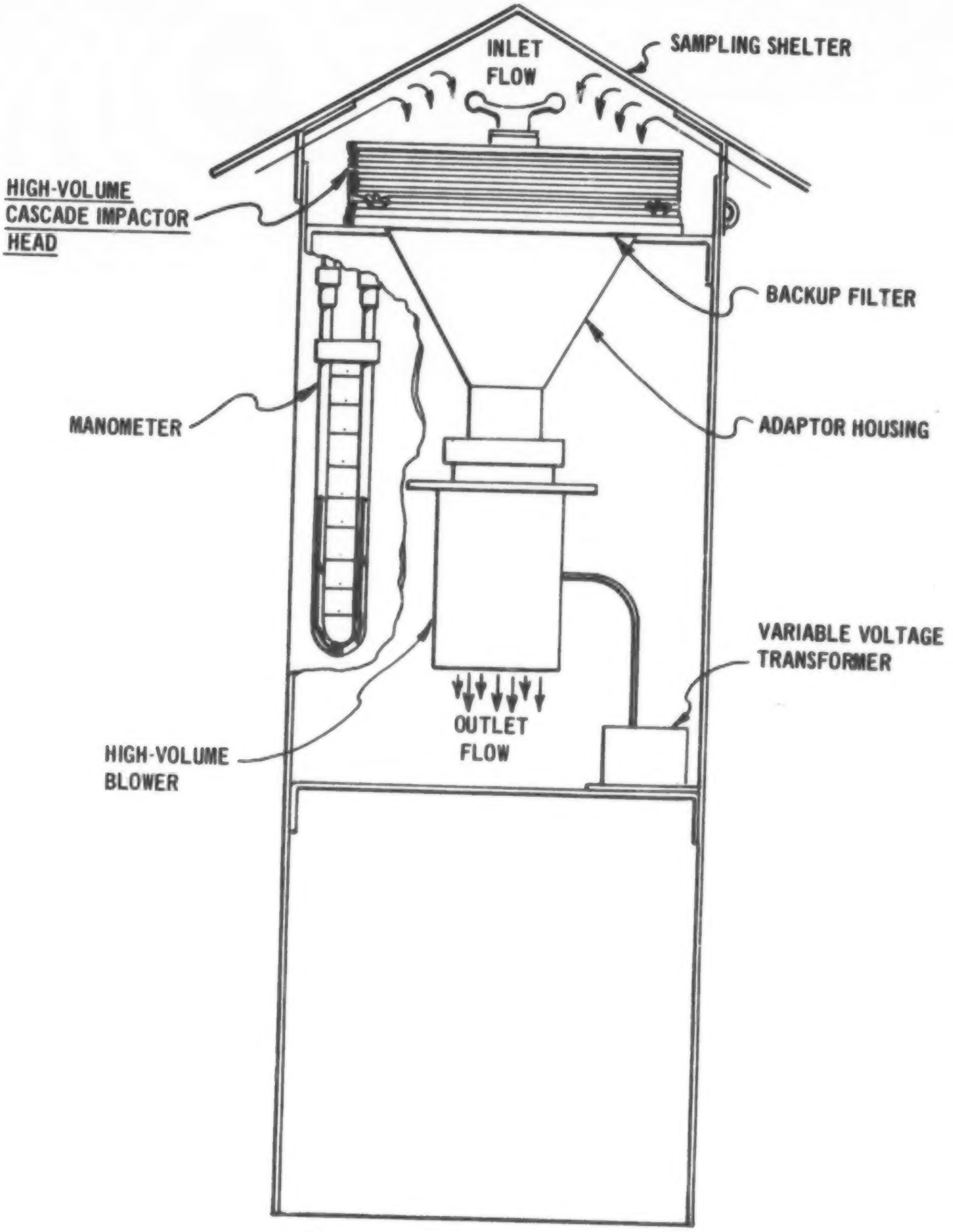


Figure A.8. Cascade impactor.

*Range, Sensitivity, and Interferences*

For 24-hour sampling, the instrument can accurately measure ambient concentrations down to 10 $\mu g/m^3$. Weights on each filter average between 5 and 50 mg and are determined on a balance with a sensitivity of 0.01 mg. Overloading and subsequent flow-rate drop are eliminated for ambient air sampling since only the particles <0.93 $\mu m$ impinge on collection media through which the airflow must pass. Sulfate formation on certain types of filter media may cause erroneous elevated mass numbers.[21]

Correlations between duplicate instruments and with the standard high-volume sampler show a high degree of correlation.[18,22]

### *Particle Size Fractionation*

The impactor fractionates particles into five aerodynamic size ranges:

| | |
|---|---|
| Stage 1 | 5.5 µm and greater |
| Stage 2 | 2.40 to 5.5 µm |
| Stage 3 | 1.75 to 2.40 µm |
| Stage 4 | 0.93 to 1.75 µm |
| Stage 5 | 0 to 0.93 µm |

The instrument has been calibrated for particle size cutoff by the Environmental Protection Agency.[21]

### *Airflow Calibration*

Each high-volume sampler head should be initially calibrated and a pressure drop versus altitude curve drawn for each serial numbered head. This pressure drop is determined with the high-volume sampler head installed and a backup filter (glass fiber type A) placed on the 20- by 25-cm filter holder. Any other type backup filter would require a new calibration in the field. A small orifice of 0.914 mm (0.036 inch) is drilled in the brass fitting (screwed in the interface plate) where a flowmeter (rotometer) may be attached in place of the manometer. Since the orifice only measures partial flow, the flowmeter must be originally calibrated with the complete high-volume air sampler using a standard high-volume rootsmeter or equivalent. The method of calibration is very similar to that specified in the Federal Register for calibrating the standard high-volume samplers.[8]

### *Care and Grooming*

The high-volume sampler head is covered when not in use. Before use, stages are examined for foreign material in the holes by holding them up to a light. With proper operation, handling, and care the holes should not become plugged. Normal skin oils can cause partial plugging of the smaller holes if hands or fingers are brushed across the plates. When necessary, the jet stages are cleaned in an ultrasonic bath with a suitable solvent or carefully washed in a detergent water solution and rinsed and dried thoroughly.

The neoprene gaskets are also cleaned periodically, as required, in plain water and dried thoroughly before use. If necessary, the gaskets may be dusted with talc or baby powder after cleaning to speed up drying time. Any dampness will cause the collection paper to adhere to the gaskets.

### *Component Part Description and Function*

The major component parts of the high-volume air sampler head may be divided into nine sections:

1. Jet plates – There are five jet plates ranging in thickness from 6.35 to 1.25 mm (0.25 to 0.050 inch).
2. Interface adapter plate – This plate interfaces between the four circular plates and the standard high-volume sampler.
3. Gasket-spacer – There are five 6.35-mm-(0.25-inch-) thick neoprene gasket-spacers to separate the plates. In damp weather or high humidity, the gasket-spacer may show a tendency to adhere to the collection paper. A slight amount of dusting talc or baby powder on the top and bottom side of the gasket and careful handling of the perforated collection papers will minimize this problem.
4. Speedball handle – This handle is designed to convenience tightening down the head by hand and also carrying the head from the field to the lab. Two to three complete turns should be sufficient to seal the plates and gaskets. While air is being pulled through the sampler, it can be determined if the unit is sealed by pressing both sides of the plates down by hand and observing any change in pressure drop on the manometer.
5. Perforated collection paper – There are two configurations of specially designed, cut and perforated collection paper. Configuration I holes correspond to the jets of plates 2 and 4; Configuration II holes correspond to jets of plates 3 and 5.
6. Interface gasket – This part seals the interface plate to the high-volume filter holder.
7. Backup filter assembly – The 20- by 25-cm backup filter is located below the interface plate (standard high-volume filter holder).
8. Manometer – A manometer is provided to accurately set the unit for an equivalent pressure drop corresponding to 565 liters/min. The scale is calibrated with red gage oil to correspond with centimeters of water.
9. Variable voltage transformer – Some type of variable voltage transformer is required to properly adjust the flow rate (measured by the pressure drop across the head with a manometer and referenced to a calibration curve furnished with each serial numbered head).

*Sample Preparation and Analysis*

Sheets of glass fiber filter medium are used for collecting the particles. They are equilibrated in an environmentally controlled chamber for 24 hours, weighed, and loaded into the cascade impactor. The impactor is shipped to the sampling site, operated for 24 hours, and returned to the laboratory. Upon arrival, the exposed collection medium is equilibrated again for 24 hours and then reweighed. Particle mass for each size fraction is determined and data are expressed in micrograms of particulate matter per cubic meter of air sampled.

## Dustfall Determination (Settleable Particulate Method)[23]

*Principle and Applicability*

The dustfall bucket is an open-topped cylindrical container used to collect the larger and more dense fractions of particulate atmospheric pollutants that fall out of suspension under the normal force of gravity. Made of durable, nonreactive polyethylene and supplied with a press-on lid, the entire unit is suitable for shipment. The dustfall bucket is 20 cm (8-1/4 inches) high and 30 cm (7-1/2 inches) in diameter at the top, with a slight taper to the bottom. It has a capacity of approximately 5 liters (5 quarts). For sampling, a clean, dry bucket is suspended on an aluminum mast at a height of at least 1.8 meters (6 feet) above ground and is allowed to remain for 1 month. After sampling, the bucket is returned to the laboratory, where the contents of the bucket are emptied into a glass beaker, any water is evaporated, and the weight of the remaining matter is determined. Residue is analyzed chemically for trace metals.[24] Settleable particles are defined as any particles, liquid or solid, small enough to pass through a 1-mm screen and large enough to settle in the collector.

*Interferences*

Care must be taken to avoid matter from trees, bird droppings, and other such deposits. Loss of material from the collector by action of the wind must be prevented.

*Analysis Apparatus*

The following equipment is required for the analysis:

1. Sieve–20 mesh, brass.
2. Analytical balance–160-gram capacity, 1-mg sensitivity.
3. Drying oven–capable of maintaining oven temperature of 105 ± 2 °C.
4. Desiccator.
5. Steam bath (or water bath).
6. Instrumentation for trace metal analysis.

*Procedure*

*Dustfall*–The sample collected in the dustfall bucket (or other suitable container) is transferred through a 20-mesh sieve into a 2-liter beaker. Sufficient distilled water is added to the bucket to ensure complete transfer of the contents. The sample is evaporated to low volume on a steam bath, then quantitatively transferred to a previously dried (at 150 °C) and tared 100-ml beaker. Volume is further reduced by evaporation on the steam bath, then the sample is dried in an oven held at 105 °C. After being cooled in a desiccator, the sample is weighed to the nearest milligram.

Dustfall is expressed as grams per square meter per month, determined as follows:

$$D = \frac{W_d}{A_j} \times \frac{30}{t} \qquad (A.14)$$

where

$D$ = dustfall, $g/m^2/mo$

$W_d$ = weight of total dustfall, grams

$A_j$ = area of top of collecting jar ($0.028\ m^2$ for standard bucket), $m^2$

$t$ = sampling period, days

The term 30/t is used to standardize the measurement to a 30-day month. The sample is normally collected over a calendar month (28 to 31 days).

*Trace Metals*–A suitable volume of nitric acid ($HNO_3$) diluted with an equal volume of water, is added to the dustfall sample after dustfall has been determined. The beaker containing the sample is then heated (below the boiling point) for about 1 hour to solubilize the trace metals. The sample is then concentrated to a convenient volume to remove excess acid and facilitate transfer.

The sample is decanted into a centrifuge tube. The beaker is rinsed with distilled water to assure complete transfer, and additional water is added to bring the sample to a known, convenient volume. Then the sample is centrifuged to remove solids. The remaining solution is analyzed on an atomic absorption instrument or other instrumentation using a reagent blank prepared in the same manner as the sample. Standard curves for each metal are prepared by plotting absorbance versus metal concentration in parts per million by weight for each metal at the appropriate wave length. (Development of x-ray fluorescence methods offers much promise for future methods of analysis.)

The weight of each trace metal in the dustfall sample can then be calculated as follows:

$$W_m = \frac{C_m V_s F}{10^6 A_j} \times \frac{30}{t} \qquad \text{(A.15)}$$

where

$W_m$ = weight of metal, mg/m$^2$/ml
$C_m$ = concentration of metal in sample, ppm
$V_s$ = volume of sample, ml
$F$ = dilution factor, mg/ml
$A_j$ = area of top of collecting jar, m$^2$
$t$ = sampling period, days

## QUALITY CONTROL

In order to understand the CHESS quality control program, it is essential to understand how the quality control operation fits into the overall operation of the CHESS air monitoring program. A schematic diagram of the overall CHESS operational plan is shown in Figure A.9. The rectangles indicate normal routine functional operations. The circles indicate operations especially designed to provide quality control. Each of the quality control points has been assigned a number to facilitate the system description.

### Materials and Equipment Control

All incoming materials, such as chemicals, filter papers, etc., are routinely inspected to ensure that they meet specifications. Before these materials are used in the CHESS operational areas, scheduled checks are made at quality control point 1. Chemical reagents are prepared for gas bubbler tubes in the chemistry laboratory. These reagents are periodically checked at quality control point 2 to ensure uniformity and absence of any systematic drifts. After this operation, the filters and reagent tubes are mailed to the CHESS operational areas.

All incoming instrumentation and equipment undergo an incoming inspection to ensure that specifications are met. Before this equipment is used in the CHESS operational areas, periodic checks are performed at quality control point 3. Some equipment, such as a high-volume sampler motor, must be attached to other assemblies before the complete unit is functional. In this case, the entire unit may require calibration. At quality control point 4, periodic checks of the fitness of the assembly and the accuracy of the calibrations are performed. The equipment is then ready to be mailed to CHESS operational areas.

The CHESS area station operators are trained by Environmental Protection Agency personnel to ensure uniformity of field procedures. The operators change bubbler tubes and filter paper, adjust timing of samplers, record both start and stop airflows, record both start and stop clock times, fill out site identification information, note special comments, perform routine simple maintenance, and phone CHESS area engineers if an emergency arises. The CHESS area engineer ensures that emergency situations are solved in a timely manner.

Another quality control feature of the CHESS air monitoring program is the use of systematic duplication of instrumentation. Replicate samples are obtained and analyzed in order to determine the reproducibility of individual instruments and an estimate of the range associated with individual measurement techniques. The above controls are represented by quality control point 5.

Routine preventive maintenance is used to minimize field equipment failures. Field equipment is returned in accordance with a preventive maintenance schedule from the CHESS area to the CHESS central maintenance area. This equipment, along with equipment that has failed in the field, is centrally repaired, refurbished, and calibrated. Scheduled checks of this equipment are made at quality control point 6 before this equipment is returned to a CHESS operational area.

### Equipment Early Warning System

Upon receipt of field samples, CHESS central personnel immediately examine the field data cards to determine any abnormality in the performance of the CHESS equipment or the CHESS area operators.

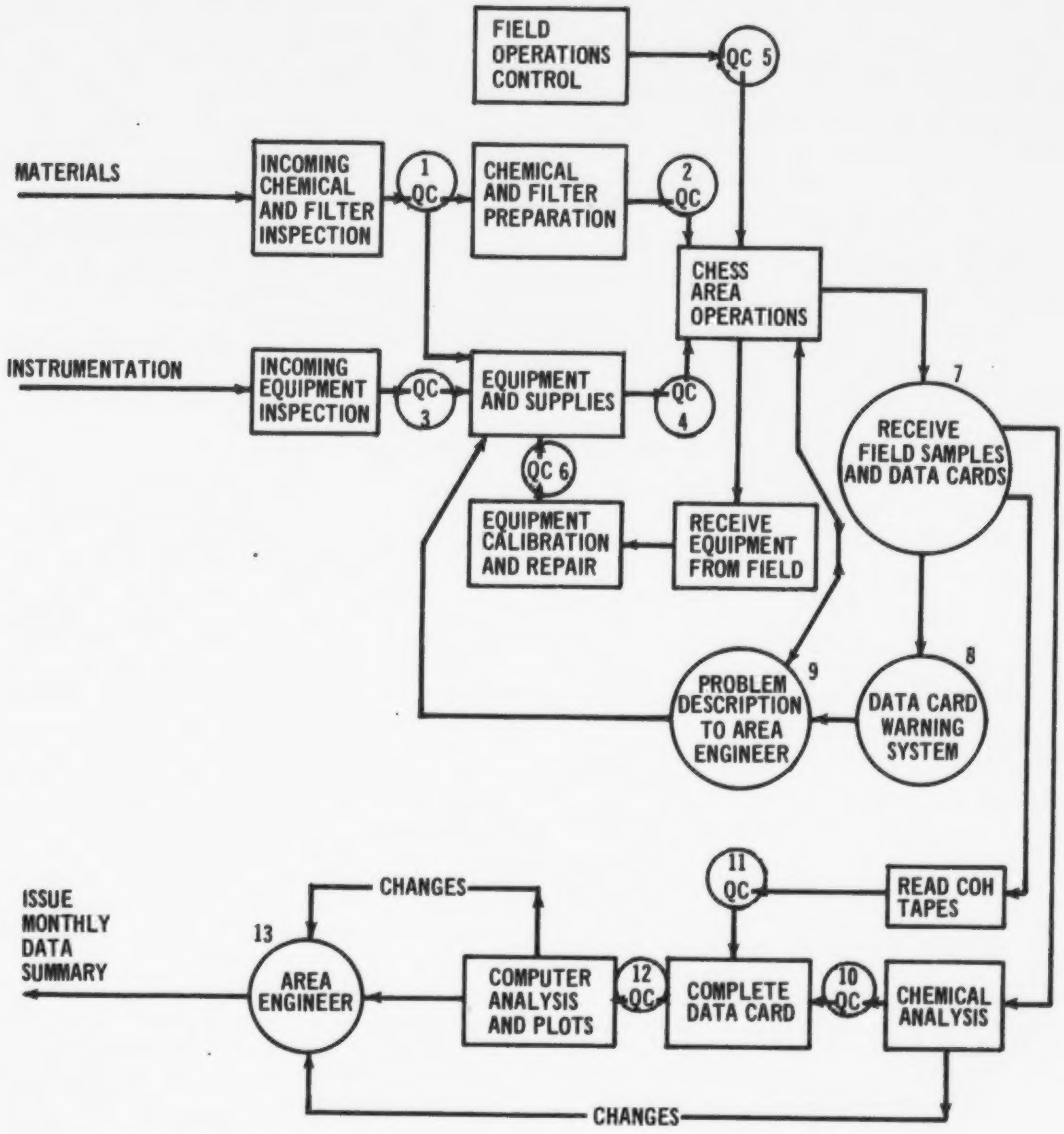

FIELD OPERATIONS CONTROL
QC 5
MATERIALS
INCOMING CHEMICAL AND FILTER INSPECTION
1 QC
CHEMICAL AND FILTER PREPARATION
2 QC
CHESS AREA OPERATIONS
INSTRUMENTATION
INCOMING EQUIPMENT INSPECTION
QC 3
EQUIPMENT AND SUPPLIES
QC 4
QC 6
EQUIPMENT CALIBRATION AND REPAIR
RECEIVE EQUIPMENT FROM FIELD
7
RECEIVE FIELD SAMPLES AND DATA CARDS
8
DATA CARD WARNING SYSTEM
9
PROBLEM DESCRIPTION TO AREA ENGINEER
11 QC
READ COH TAPES
ISSUE MONTHLY DATA SUMMARY
CHANGES
13
AREA ENGINEER
COMPUTER ANALYSIS AND PLOTS
12 QC
COMPLETE DATA CARD
10 QC
CHEMICAL ANALYSIS
CHANGES

Figure A.9. CHESS quality control.

This operation is represented by quality control point 7. The area engineer is quickly notified of discrepancies by means of the data card warning system, which is represented as quality control point 8.

Typical problems that can be identified by this system include:

1. Deterioration of routeman's performance.
2. Leaks in bubbler system.
3. Need for replacement of parts.
4. Need for periodic site inspection.
5. Invalid samples.

The area engineer resolves the particular problem with the CHESS area operator and sends out appropriate replacement items. This function is represented as quality control point 9.

## Chemical Analysis

The CHESS area specimens are analyzed in the chemistry laboratory, following documented quality control procedures.[25] The analyses follow Federal Register procedures where possible. Systematic checks of procedures are made by analyzing standard samples that contain known amounts of a specific air pollutant. Data are calculated in the lab before release in order to ensure their validity. A visual inspection of each data card is made in order to ensure completeness. These operations are represented by quality control point 10. Data, such as COH levels, not requiring measurement at the laboratory are checked at control point 11, and data cards are prepared.

## Computer Analysis

The field data cards are sent to the computer group for computer card punching. These cards are inspected for card punch errors before they are analyzed by the computer. This inspection is represented by quality control point 12.

The data are analyzed by computer according to an analysis protocol. Examples of monthly averages for given CHESS monitoring stations are shown in Figure A.10. These averages provide a basis of

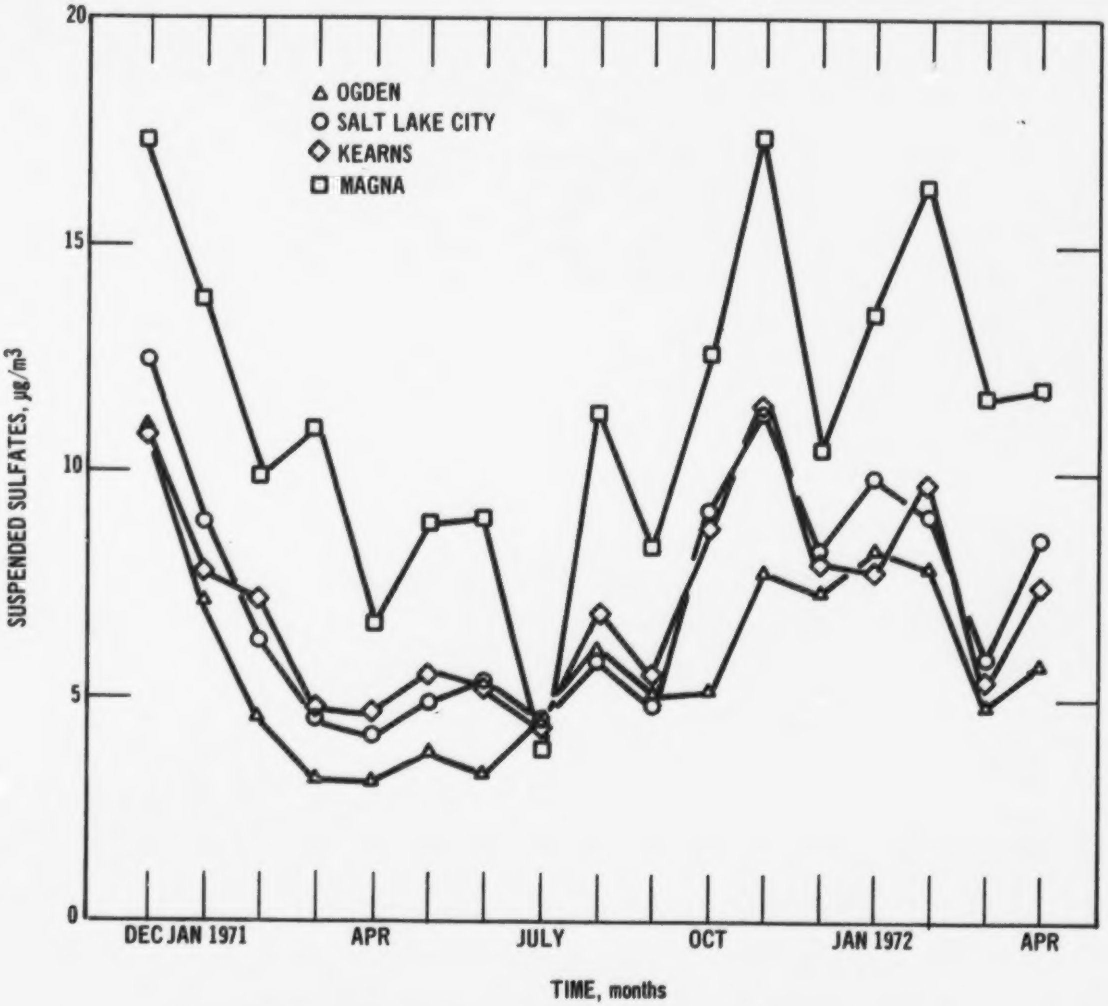
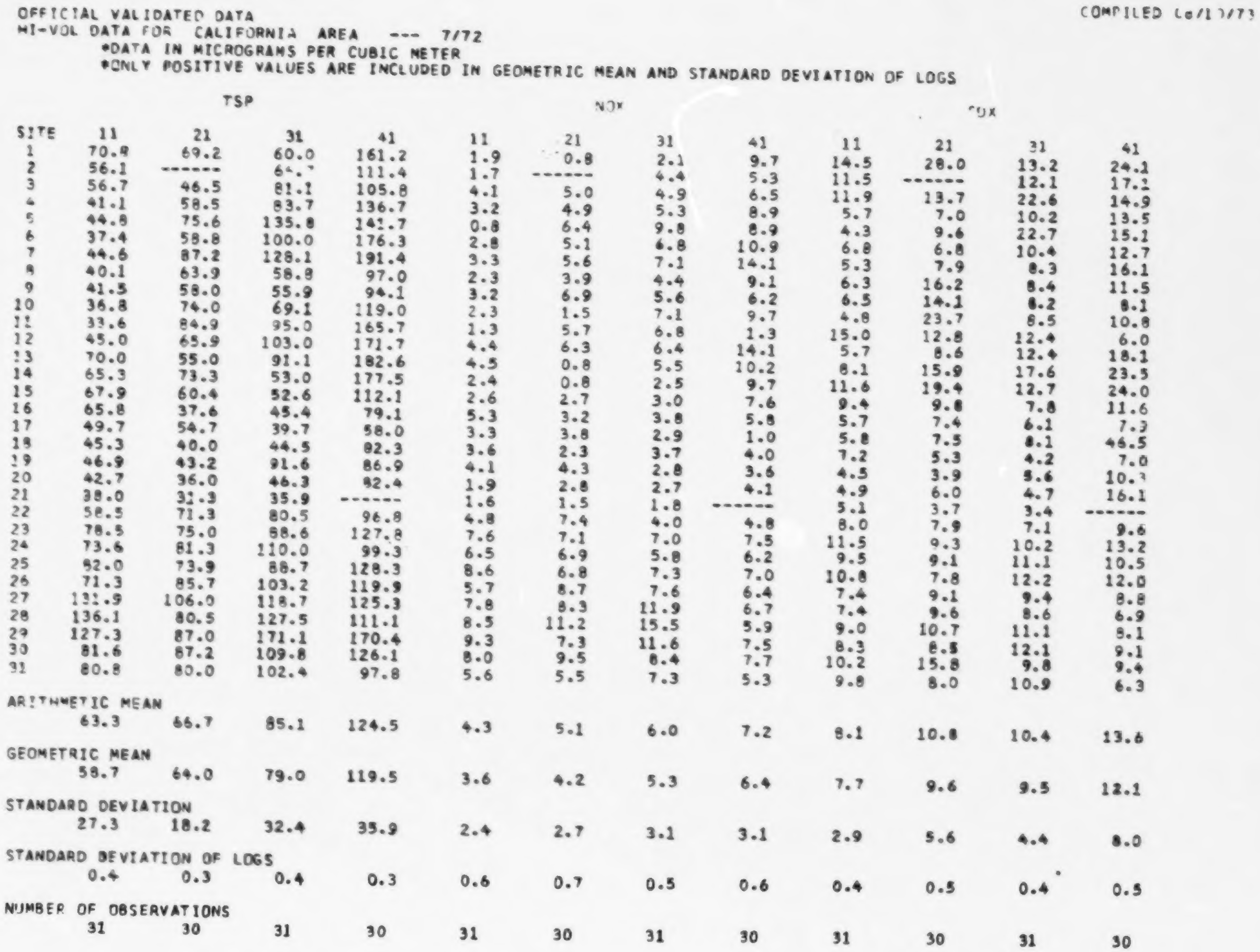


Figure A. 10. Drawing based on graphical output of monthly data.



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OFFICIAL VALIDATED DATA                                                                              COMPILED 6/1[illegible]/73
HI-VOL DATA FOR  CALIFORNIA  AREA   ---  7/72
         *DATA IN MICROGRAMS PER CUBIC METER
         *ONLY POSITIVE VALUES ARE INCLUDED IN GEOMETRIC MEAN AND STANDARD DEVIATION OF LOGS

                      TSP                                    NOX                                   SOX

SITE    11        21        31        41         11       21       31       41       11       21       31       41
  1    70.9      69.2      60.0     161.2       1.9      0.8      2.1      9.7     14.5     28.0     13.2     24.1
  2    56.1    ------      6[illegible]     111.4       1.7   ------      4.4      5.3     11.5   ------     12.1     17.1
  3    56.7      46.5      81.1     105.8       4.1      5.0      4.9      6.5     11.9     13.7     22.6     14.9
  4    41.1      58.5      83.7     136.7       3.2      4.9      5.3      8.9      5.7      7.0     10.2     13.5
  5    44.8      75.6     135.8     141.7       0.8      6.4      9.8      8.9      4.3      9.6     22.7     15.1
  6    37.4      58.8     100.0     176.3       2.8      5.1      6.8     10.9      6.8      6.8     10.4     12.7
  7    44.6      87.2     128.1     191.4       3.3      5.6      7.1     14.1      5.3      7.9      8.3     16.1
  8    40.1      63.9      58.8      97.0       2.3      3.9      4.4      9.1      6.3     16.2      8.4     11.5
  9    41.5      58.0      55.9      94.1       3.2      6.9      5.6      6.2      6.5     14.1      8.2      8.1
 10    36.8      74.0      69.1     119.0       2.3      1.5      7.1      9.7      4.8     23.7      8.5     10.8
 11    33.6      84.9      95.0     165.7       1.3      5.7      6.8      1.3     15.0     12.8     12.4      6.0
 12    45.0      65.9     103.0     171.7       4.4      6.3      6.4     14.1      5.7      8.6     12.4     18.1
 13    70.0      55.0      91.1     182.6       4.5      0.8      5.5     10.2      8.1     15.9     17.6     23.5
 14    65.3      73.3      53.0     177.5       2.4      0.8      2.5      9.7     11.6     19.4     12.7     24.0
 15    67.9      60.4      52.6     112.1       2.6      2.7      3.0      7.6      9.4      9.8      7.8     11.6
 16    65.8      37.6      45.4      79.1       5.3      3.2      3.8      5.8      5.7      7.4      6.1      7.3
 17    49.7      54.7      39.7      58.0       3.3      3.8      2.9      1.0      5.8      7.5      8.1     46.5
 18    45.3      40.0      44.5      82.3       3.6      2.3      3.7      4.0      7.2      5.3      4.2      7.0
 19    46.9      43.2      91.6      86.9       4.1      4.3      2.8      3.6      4.5      3.9      5.6     10.3
 20    42.7      36.0      46.3      82.4       1.9      2.8      2.7      4.1      4.9      6.0      4.7     16.1
 21    38.0      31.3      35.9    ------       1.6      1.5      1.8   ------      5.1      3.7      3.4   ------
 22    58.5      71.3      80.5      96.8       4.8      7.4      4.0      4.8      8.0      7.9      7.1      9.6
 23    78.5      75.0      88.6     127.8       7.6      7.1      7.0      7.5     11.5      9.3     10.2     13.2
 24    73.6      81.3     110.0      99.3       6.5      6.9      5.8      6.2      9.5      9.1     11.1     10.5
 25    82.0      73.9      88.7     128.3       8.6      6.8      7.3      7.0     10.8      7.8     12.2     12.0
 26    71.3      85.7     103.2     119.9       5.7      8.7      7.6      6.4      7.4      9.1      9.4      8.8
 27   131.9     106.0     118.7     125.3       7.8      8.3     11.9      6.7      7.4      9.6      8.6      6.9
 28   136.1      80.5     127.5     111.1       8.5     11.2     15.5      5.9      9.0     10.7     11.1      8.1
 29   127.3      87.0     171.1     170.4       9.3      7.3     11.6      7.5      8.3      8.5     12.1      9.1
 30    81.6      87.2     109.8     126.1       8.0      9.5      8.4      7.7     10.2     15.8      9.8      9.4
 31    80.8      80.0     102.4      97.8       5.6      5.5      7.3      5.3      9.8      8.0     10.9      6.3

ARITHMETIC MEAN
       63.3      66.7      85.1     124.5       4.3      5.1      6.0      7.2      8.1     10.8     10.4     13.6

GEOMETRIC MEAN
       58.7      64.0      79.0     119.5       3.6      4.2      5.3      6.4      7.7      9.6      9.5     12.1

STANDARD DEVIATION
       27.3      18.2      32.4      35.9       2.4      2.7      3.1      3.1      2.9      5.6      4.4      8.0

STANDARD DEVIATION OF LOGS
        0.4       0.3       0.4       0.3       0.6      0.7      0.5      0.6      0.4      0.5      0.4      0.5

NUMBER OF OBSERVATIONS
        31        30        31        30        31       30       31       30       31       30       31       30
```

Figure A.11. Example of tabular computer output of monthly data.

comparison for individual data points. If a point appears to be in error, both the chemistry laboratory and the computer group are notified as required. The above functions are represented by quality control point 13. After the aerometry field data have been successfully analyzed, they are compiled by the area engineer and issued as a monthly data summary. A typical monthly summary is shown in Figure A.11.

## PRECISION OF MEASUREMENT

In order to determine the precision of the air monitoring techniques used in the CHESS program, a series of special studies were initiated. The studies were performed in four cities in the southeastern United States--Birmingham, Alabama, Charlotte and Greensboro, North Carolina, and Chattanooga, Tennessee. Duplicate air monitoring equipment was installed in selected sites in order to determine the daily range and daily percentage error for a given air measurement.

The range is determined by taking the absolute value of the difference between the pollutant concentration measured with the original equipment and that measured with the duplicate equipment. An estimate of the daily error between original and duplicate is made by dividing one-half the range by the average value.

$$\pm\%E = \frac{(C_o - C_d)/2}{(C_o + C_d)/2} \times 100 = \frac{C_o - C_d}{C_o + C_d} \times 100 \qquad \text{(A.16)}$$

where

$\%E$ = daily percentage error
$C_o$ = daily concentration from original equipment
$C_d$ = daily concentration from duplicate equipment

The daily percentage errors over the period of time for particular studies are tabulated in the order of increasing error, for example, 2, 5, 9 percent, etc. The rank of these percentage errors is listed next to a given percentage. An estimate of cumulative frequency is made by dividing the rank of a given percentage error (n) by the total number of duplicate samples (m), plus one.

$$CF = \frac{n}{m + 1} \qquad \text{(A.17)}$$

The estimate of the error at the 50 percent cumulative frequency is obtained by determining the percentage error value that corresponds to a cumulative frequency of 50 percent. The 50 percent frequency is chosen because 50 percent of the data have errors that are smaller than the value, and likewise 50 percent are greater.

### Sulfur Dioxide

A study of the precision of sulfur dioxide measurements was made in Birmingham over a 20-month period. The results of this study are shown in Figure A.12. The 50 percent cumulative frequency corresponds to an error of ±18 percent.

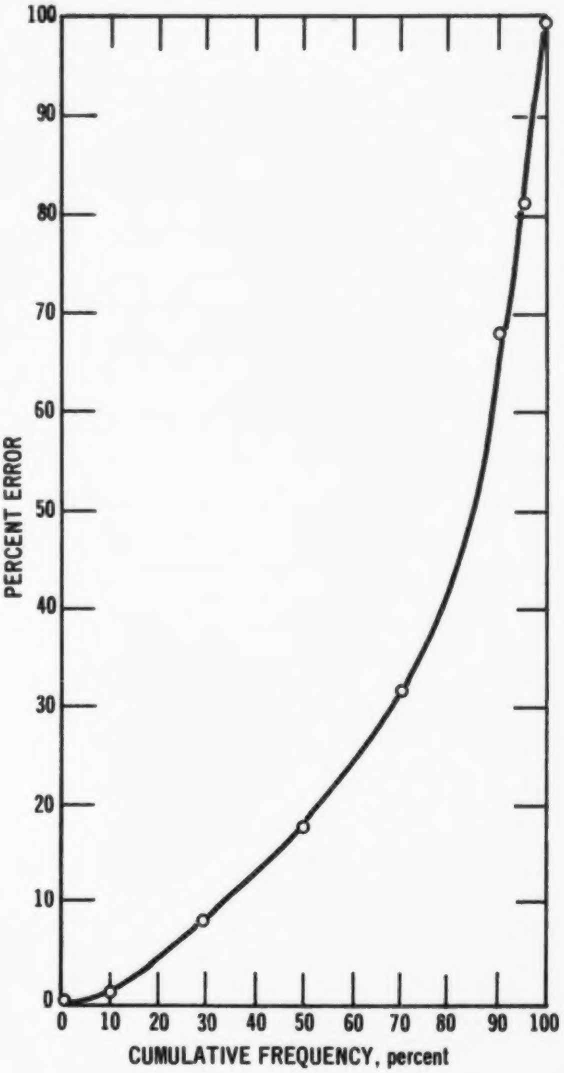

Figure A.12 Precision of CHESS sulfur dioxide measurements (Birmingham, October 1969-May 1971).

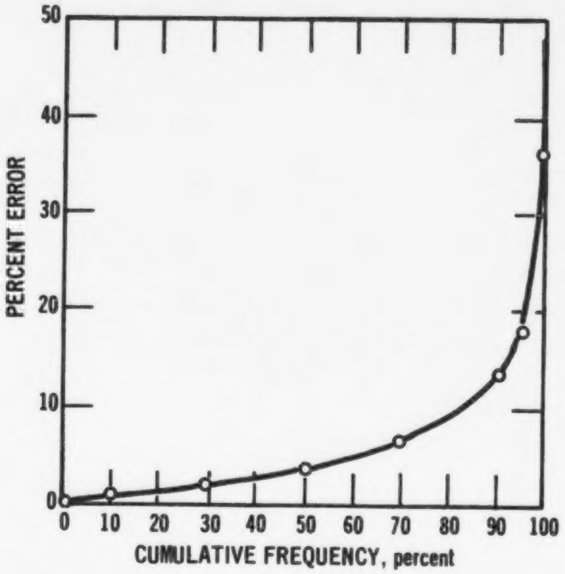

Figure A.13. Precision of CHESS particulate measurements (Birmingham, Charlotte, and Greensboro, May 1971-April 1972).

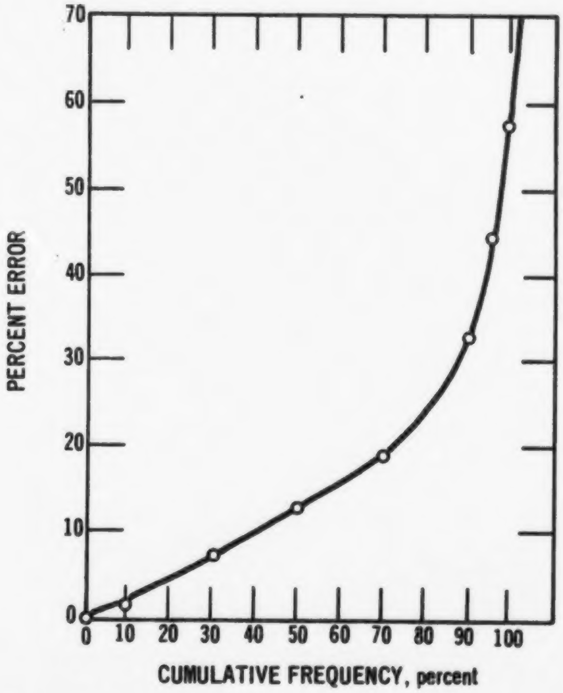

Figure A.14. Precision of CHESS nitrogen dioxide measurements (Chattanooga, September 1971-March 1972).

### Total Suspended Particulate

A similar 12-month study was performed in three southeastern CHESS cities (Birmingham, Charlotte, and Greensboro) to determine the precision of total suspended particulate measurements. A graph of the data from this study is shown in Figure A.13. The 50 percent cumulative frequency corresponds to an error of 4 percent.

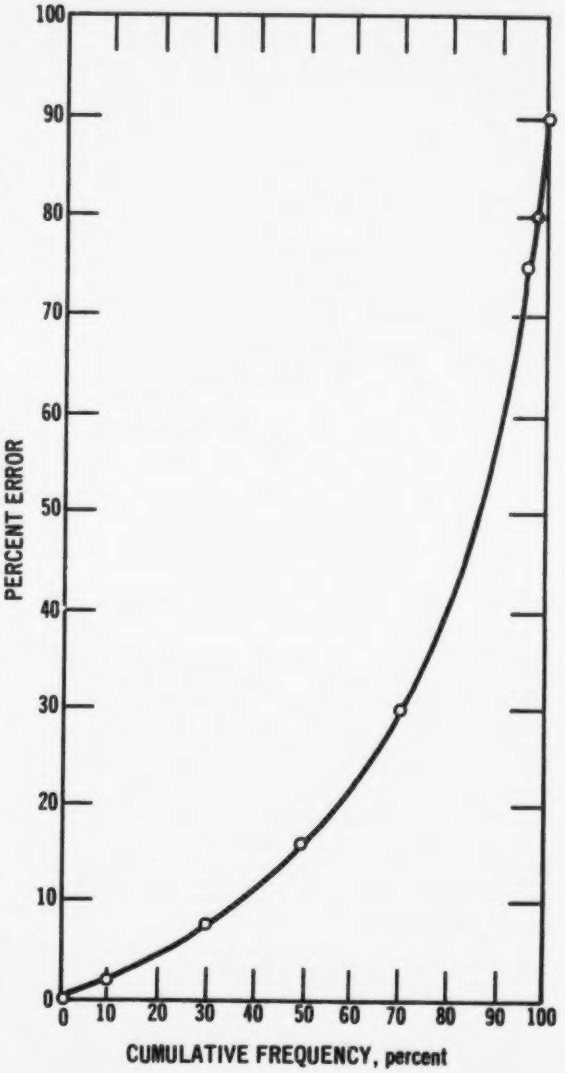

Figure A.15. Precision of CHESS dustfall measurements (Birmingham, Charlotte, and Greensboro, October 1969-March 1972)

### Nitrogen Dioxide

A 7-month study of the precision of nitrogen dioxide measurements was performed in Chattanooga. A graphical presentation of the percent error frequency distribution is shown in Figure A.14. The 50 percent cumulative frequency corresponds to an error of ±13 percent.

### Total Dustfall

An 18-month study of the precision of CHESS monthly dustfall measurements was performed in three southeastern CHESS cities (Birmingham, Charlotte, and Greensboro). The data obtained in this study are summarized in Figure A.15. The 50 percent cumulative frequency corresponds to an error of 16 percent.

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# APPENDIX B:
# NASN LABORATORY METHODOLOGY

In estimating human exposure to air pollutants for Community Health and Environmental Surveillance System (CHESS) studies, data from CHESS sites are supplemented by available data from other Federal, state, and local sampling sites. One of the most frequently available sources of such supplemental data is the Environmental Protection Agency's National Air Sampling Network. NASN laboratory methodology is therefore described in this appendix.

## SUSPENDED PARTICULATE MATTER

The weight or mass of particulate matter per volume of air is usually determined by drawing air through a weighed filter with a high-volume air sampler, and then reweighing the soiled filter. Particular laboratory procedures, however, may differ from one laboratory to another. The following procedure is used by the Division of Air Surveillance for all sampling conducted by the NASN.

Each city's high-volume sampler is located in the central business-commercial district at a site that is, as nearly as practical, comparable with the corresponding sites in other cities. In such a location, measured concentrations are among the higher concentrations found in the city, and therefore cannot be interpreted as the citywide average.

The high-volume samplers, operating at from 1130 to 1700 liters/min (40 to 60 $ft^3$/min), collect particles from about 2200 $m^3$ of air during the 24-hour sampling period. The NASN stations are operated during 25 randomly selected days per year. The filters used are 20- by 25-cm (8- by 10-inch) fiber filters selected for low and uniform background concentrations of those substances to be measured.

To eliminate any filters having pinholes or other flaws that could affect airflow, the filters are screened on a light table. Prior to weighing, they are equilibrated for 24 hours at 24 °C and not more than 50 percent relative humidity. Because any crease in a filter can seriously affect the airflow, filters are weighed with a balance that permits weighing without bending.

The filters are then distributed unfolded to the cooperating local agencies. After sampling, the filters are folded in half with the collected particulate matter inside and returned to the laboratory. After a filter with the collected particulate matter is again equilibrated for at least 24 hours at 24 °C and not more than 50 percent relative humidity, it is weighed to determine the amount of particulate matter collected. The equilibration at low relative humidity prior to this weighing is crucial, because many of the compounds collected (ammonium sulfate in particular) are quite hygroscopic. Figure B.1 shows the effect of humidity on the measured weights of soiled filters from three widely differing sampling locations. The increase in weight at the higher humidities represents the amount of atmospheric moisture collected by the particulate matter on the filters.

## ORGANIC PARTICULATE MATTER

The numerous specific organic compounds that are removed by benzene include many of the manmade organic pollutants. Natural organic airborne particles, such as pollens and molds, generally are not soluble in benzene.

Benzene-soluble organics are determined by extraction of aliquots of the samples with redistilled benzene in a Soxhlet extractor for from 6 to 8 hours. This extraction removes more than 90 percent of the benzene-soluble organic materials from the sample. After extraction, the solution is concentrated and is filtered into a tared test tube. The benzene solvent is then evaporated at 60 °C, and the organic matter remaining is weighed.

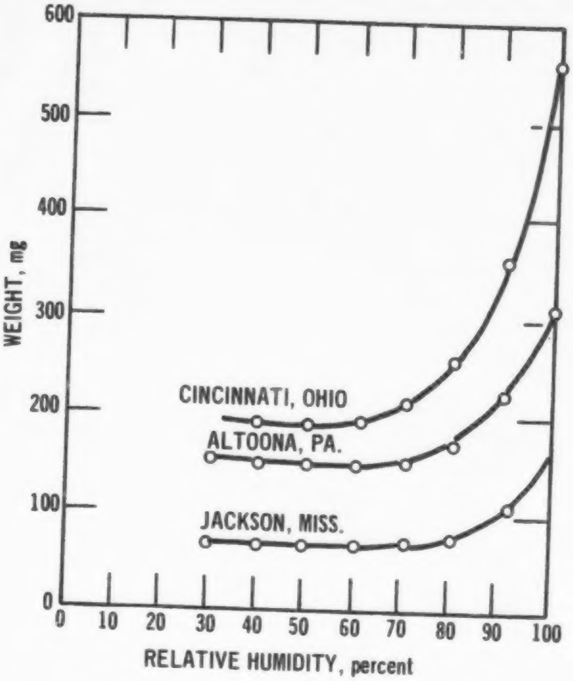

Figure B.1. Effects of relative humidity on weight of atmospheric particulates at 24°C.

The benzo(a)pyrene (BaP) component of the benzene-soluble organic residue is determined after first separating the different components by thin-layer chromatography, then removing the BaP from the thin-layer plate and thin-layer adsorbent. The BaP is then dissolved in sulfuric acid and its concentration measured by fluorescence spectroscopy.

## NONMETALLIC INORGANIC CONSTITUENTS

Ammonium, nitrate, and sulfate ions are automatically analyzed using an aqueous extract of an 8.3 percent aliquot of the particulate sample. The sample aliquot is refluxed with 50 ml of distilled water in a 125-ml flask, cooled and filtered, and then reextracted with 10 to 15 ml of water. In filtration of this extract, the filter and flasks are washed to a total volume of 50 ml of filtrate, which is then mixed and used for the three analyses.

Ammonium ion in the filtrate is reduced to nitrite by alkaline hydrazine (pH about 11). Sulfanilamide is then added to form a diazo compound, which is subsequently combined with N-1-napthylethylenediamine. The resulting compound is measured spectrophotometrically at 535 nm. This method offers distinct advantages in avoiding interferences from other water-soluble compounds found in atmospheric particulate matter.

Sulfate ion in the filtrate is determined by the methylthymol blue method, which is appropriate only with automatic analysis techniques because the methylthymol blue dye is oxidized by atmospheric oxygen. The filtrate is reacted with a reagent consisting of equal parts of methylthymol blue dye and barium chloride, kept at a pH of 2.8 to prevent the formation of a chelate complex from the dye and the barium. Any sulfate ion in the sample reacts with the barium, leaving an excess of methylthymol blue dye that is proportional to the amount of sulfate present. The pH is then raised to 12.4, at which point the barium not removed by the sulfate forms a chelate complex with the methylthymol blue dye and the excess dye turns yellow. The intensity of the yellow color is then determined colorimetrically at 480 μm.

Water-soluble fluorides are also measured using a fluoride selective electrode technique.[1] The analysis is performed on an aliquot of the same water extract prepared for the ammonium, nitrate, and sulfate analyses. A sodium citrate-carbonate buffer solution is added to adjust the pH ionic strength and control interferences from metal ions. The fluoride-selective electrode generates a potential in proportion to the fluoride activity in the sample that is measured against the potential of a standard reference electrode.

## METALS

In the metals analysis, an aliquot of the sample is ashed and then extracted with a mixture of distilled hydrochloric acid and nitric acid. The manner of ashing the sample is critical in the determination of the more volatile metals such as lead, zinc, and cadmium. At one time, samples were ashed in a muffle furnace at 500 °C, but they are now ashed in a low-temperature asher (at about 150 °C). After ashing and extraction, metals contents of urban samples are determined with an emission spectograph.

## GASES

The gas samples are gathered with a bubbler-train sampler. Air is drawn successively through a membrane filter, a manifold, a bubbler, another filter, and finally a critical orifice. The membrane prefilter removes particulate matter; the glass manifold permits the connection of up to five bubblers in parallel. The bubbler itself is filled with the appropriate absorbing reagent and is followed by a postfilter, which serves to protect the hypodermic needle critical orifice, which controls the airflow. Following sample collection, the bubbler tubes are shipped to the laboratories for analysis.

The analytical method used for sulfur dioxide is the West-Gaeke method[2] with the addition of sulfamic acid to the method modified for the AutoAnalyzer by Welch and Terry.[3] Sulfur dioxide is collected from the air sample by the complexing action of sodium tetrachloromercurate and is determined colorimetrically by measuring the red-violet color produced when a bleached rosaniline hydrochloride solution and dilute aqueous formaldehyde react with the sulfur dioxide in the collecting solution. The sulfamic acid prevents interference from nitrogen dioxide.

Nitrogen dioxide measurements are based on the Jacobs-Hochheiser and Saltzman techniques.[4,5] When the sample air containing nitrogen dioxide is drawn through a sodium hydroxide solution, a dilute solution of sodium nitrite and sodium nitrate is produced. In the laboratory, this collecting solution is reacted with solutions containing phosphoric acid, sulfanilamide, and N-1-naphthylethylenediamine dihydrochloride. The transmittance of the resulting azo dye is measured colorimetrically.

The analytical method for aldehydes is based on the fact that MBTH (3-methyl-2-benzothiazolone hydrozone hydrochloride) reacts with aliphatic aldehydes in the presence of acid ferric chloride. Aldehydes are collected by drawing air through a 0.05 percent solution of MBTH contained in a special polypropylene collection tube. The special tube is required because both the MBTH and the aldehydes are unstable when allowed to come in contact with rubber, polyethylene, or Tygon, materials that are otherwise used in the gas sampler. The sampling tubes contain 50 ml of distilled water when sent into the field. After the sample is returned to the laboratory, color is developed by the addition of ferric chloride-sulfamic acid solution, and measured colorimetrically. Results are expressed as formaldehyde.

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# APPENDIX C: QUESTIONNAIRES USED IN THE CHESS STUDIES

The following questionnaires were used for the CHESS studies covered in this report:

1. School and Family Health Questionnaire (Table C.1).
2. Acute Respiratory Disease Questionnaire (Table C.2).
3. Asthma Panel Questionnaire (Table C.3).
4. Asthma Diary (Table C.4).
5. Elderly Panel Questionnaire (Table C.5).
6. Elderly Panel Health Questionnaire (Table C.6).
7. "Well" Panel Diary (Table C.7).
8. "Heart" Panel Diary (Table C.8).
9. "Lung" Panel Diary (Table C.9).
10. "Heart and Lung" Panel Diary (Table C.10).

The School and Family Health Questionnaire (Table C.1), modified for self-administration for these studies from a standardized British Medical Research Council questionnaire for chronic respiratory disease, was distributed through schools in the Salt Lake and Rocky Mountain study communities, and a similar form was distributed through schools in the New York communities. A modification of the form was used in Chicago to obtain health and demographic information for military recruits. Information obtained from the questionnaire was used to assess the prevalence of chronic respiratory disease in adults in the Salt Lake, Rocky Mountain, and New York areas, prevalence of chronic respiratory disease among military recruits in the Chicago area, and prevalence of acute lower respiratory illness in children in the Salt Lake and Rocky Mountain areas.

The Acute Respiratory Disease Questionnaire (Table C.2) was used to obtain information on acute respiratory disease in volunteer families in the Chicago and New York communities. The forms were completed by trained interviewers on the basis of telephone calls that covered 2-week periods.

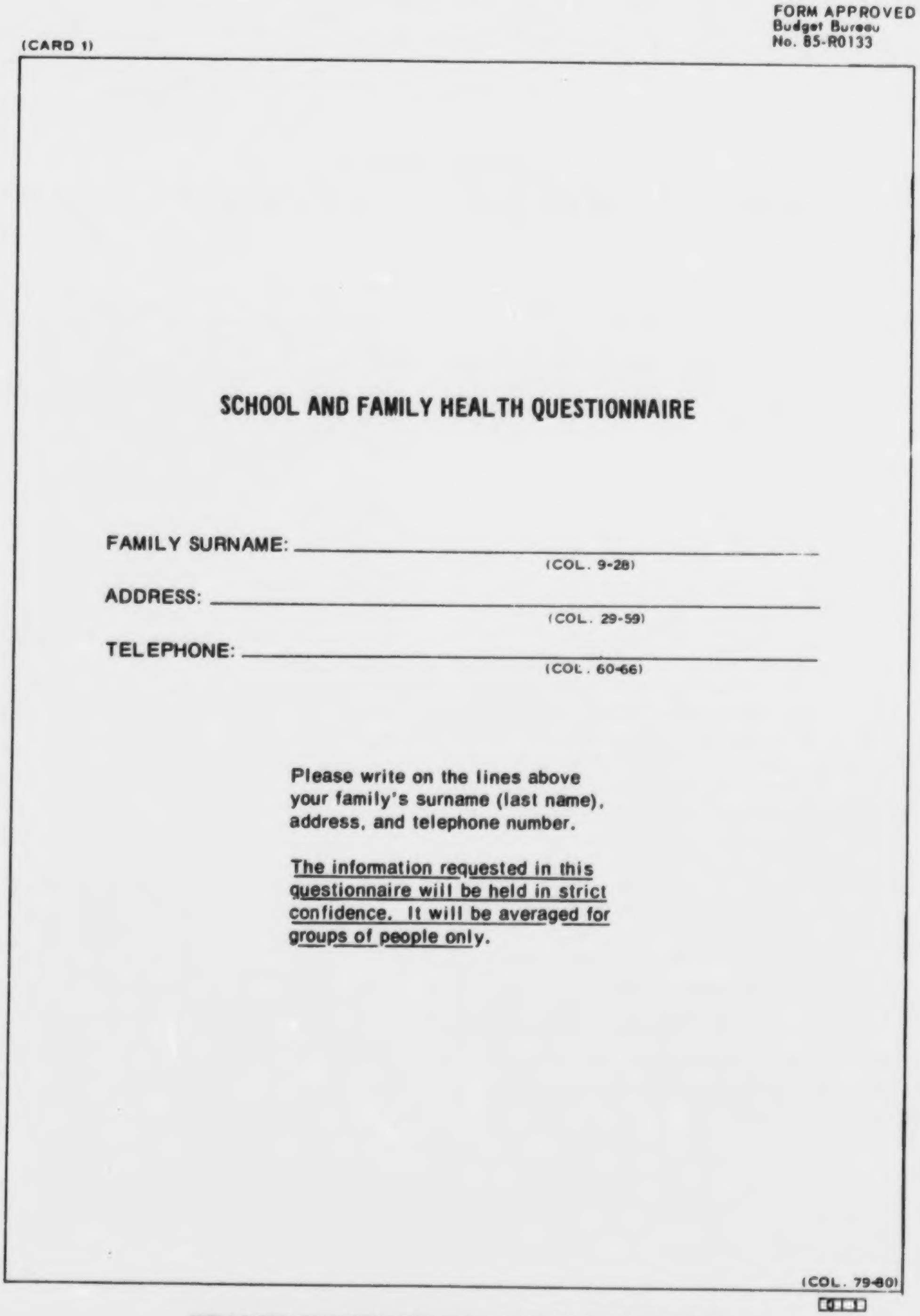
The Asthma Panel Questionnaire (Table C.3) was used to enroll and classify panelists for asthma studies in the Salt Lake and New York communities. The Asthma Diary (Table C.4) was completed by the panelists on a weekly basis.

The Elderly Panel Questionnaire and Elderly Panel Health Questionnaire (Tables C.5 and C.6) were used to enroll and classify panelists for a study of cardiopulmonary symptoms in the New York communities. On the basis of answers to questions on the health questionnaire, participants in this study were partitioned into four panels:

1. "Well" – no to questions 3, 6, 8, 9, 10, 12, 13, and 14.
2. "Heart" – yes to questions 8, 9, 10, 12, 13, or 14; no to questions 3 and 6.
3. "Lung" – yes to questions 3 or 6; no to questions 8, 9, 10, 12, 13, and 14.
4. "Heart and lung" – yes to questions 8, 9, 10, 12, 13, or 14 and yes to questions 3 or 6.

The panelist then completed weekly diaries appropriate to their respective panels (Tables C.7 to C.10).

## Table C.1. SCHOOL AND FAMILY HEALTH QUESTIONNAIRE



FORM APPROVED
Budget Bureau
No. 85-R0133

(CARD 1)

### SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

FAMILY SURNAME: ______________________________ (COL. 9-28)

ADDRESS: ______________________________ (COL. 29-59)

TELEPHONE: ______________________________ (COL. 60-66)

Please write on the lines above your family's surname (last name), address, and telephone number.

The information requested in this questionnaire will be held in strict confidence. It will be averaged for groups of people only.

(COL. 79-80)

[illegible]

## Table C.1. (continued). SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

### I. HEALTH QUESTIONS CONCERNING FATHER AND MOTHER ONLY

(CARD 2)

NOTE The questions in this section are to be answered for both parents by the mother or female guardian. Answer the questions in this section only for parents (or guardians) living in the home.

| | MOTHER (or female guardian) | | FATHER (or male guardian) | |
|---|---|---|---|---|
| | (COL. 9) | | (COL. 10) | |
| 1. Do you usually cough first thing in the morning in winter? (Count two or more coughs upon arising, or when you first go out of doors, or when you smoke the first cigarette of the day. Do not count clearing of throat.) | Yes ☐ 1 | No ☐ 2 | Yes ☐ 1 | No |
| | (COL. 11) | | (COL. 12) | |
| 2. Do you usually cough during the day or night in winter? (Do not count an occasional cough.) | Yes ☐ 1 | No ☐ 2 | Yes ☐ 1 | No ☐ 2 |
| IF YOU ANSWERED "YES" TO QUESTION 1 OR 2. PLEASE ANSWER QUESTION 3. | (COL. 13) | | (COL. 14) | |
| 3. Do you cough like this on most days or nights for as much as three months each year? | Yes ☐ 1 | No ☐ 2 | Yes ☐ 1 | No ☐ 2 |

| | MOTHER (or female guardian) | | FATHER (or male guardian) | |
|---|---|---|---|---|
| | (COL. 15) | | (COL. 16) | |
| 4. Do you usually bring up phlegm (thick fluid) from your chest first thing in the morning in winter? (Count phlegm whether swallowed or expelled, upon arising, or when you first go out of doors, or when you smoke the first cigarette of the day. Do not count phlegm from nose.) | Yes 1 | No 2 | Yes 1 | No 2 |
| | (COL. 17) | | (COL. 18) | |
| 5. Do you usually bring up phlegm from your chest during the day or night in winter? | Yes 1 | No 2 | Yes 1 | No 2 |
| IF YOU ANSWERED "YES" TO QUESTION 4 OR 5. PLEASE ANSWER QUESTION 6. | | | | |
| | (COL. 19) | | (COL. 20) | |
| 6. Do you bring up phlegm like this on most days for as much as three months each year? | Yes 1 | No 2 | Yes 1 | No 2 |
| | (COL. 21) | | (COL. 22) | |
| 7. Do you get short of breath walking on level ground at an ordinary pace? | Yes 1 | No 2 | Yes 1 | No 2 |

| | MOTHER (COL. 23) | FATHER (COL. 24) |
|---|---|---|
| 8. How many cigarettes do you usually smoke now? | | |
| Never smoked | 1 | |
| Ex-smoker | 2 | |
| Less than 1/2 pack per day (1-5 cigarettes) | 3 | |
| About 1/2 pack per day (6-14 cigarettes) | 4 | |
| About 1 pack per day (15-25 cigarettes) | | |
| About 1-1/2 packs per day (26-34 cigarettes) | | |
| About 2 or more packs per day (35 or more cigarettes) | | |

## Table C.1. (continued). SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

**FATHER-MOTHER HEALTH QUESTIONS CONTINUED**

| | MOTHER (or female guardian) (COL. 25) | FATHER (or male guardian) (COL. 26) |
|---|---|---|
| 9. At your job, are you now or have you been frequently exposed to irritating smoke, dust, or fumes? (Do not include neighborhood or home exposures.) | Yes ☐ 1 No ☐ 2 | Yes ☐ 1 No ☐ 2 |
| IF YOU ANSWERED "NO" TO QUESTION 9, SKIP THE NEXT THREE QUESTIONS BELOW. | | |
| 9a. If the answer to question 9 is "yes," what kind of irritant were you exposed to? (For example: coal dust, cutting oils, asbestos, mine dust, smelter fumes, raw cotton dust.) | ____________<br>____________ | ____________<br>____________ |
| 9b. If the answer to question 9 is "yes," what kind of work did you perform in this job? (For example: miner, maintenance, assembly line, supervisor.) | ____________<br>____________ | ____________<br>____________ |
| 9c. If the answer to question 9 is "yes," how long were you exposed? | (COL. 27) | (COL. 28) |
| Less than 1 year | ☐ 1 | ☐ 1 |
| 1 to 5 years | ☐ 2 | ☐ 2 |
| 6 to 10 years | ☐ 3 | ☐ 3 |
| More than 10 years | ☐ 4 | ☐ 4 |
| DO NOT MARK THESE BOXES | (COL. 29)<br>Yes ☐ 1 No ☐ 2 | (COL. 30)<br>Yes ☐ 1 No ☐ 2 |

COL. 79-80 [0 4]

5. How long has the family lived in your present city or town? (Check one box only.)

(COL. 13-14)

| | | |
|---|---|---|
| ☐ 01 Less than 1 year | ☐ 06 5 years | ☐ 11 10 years |
| ☐ 02 1 year | ☐ 07 6 years | ☐ 12 11 years |
| ☐ 03 2 years | ☐ 08 7 years | ☐ 13 12 years or more |
| ☐ 04 3 years | ☐ 09 8 years | |
| ☐ 05 4 years | ☐ 10 9 years | |

6. How many rooms are there in your living quarters? (Do not count bathrooms, porches, balconies, foyers, halls or half-rooms.) (Check one box only.)

(COL. 15)

| | |
|---|---|
| ☐ 1 One | ☐ 6 Six |
| ☐ 2 Two | ☐ 7 Seven |
| ☐ 3 Three | ☐ 8 Eight |
| ☐ 4 Four | ☐ 9 Nine or more |
| ☐ 5 Five | |

7. Do you have air conditioning in your living quarters? (Check one box only.)

(COL. 16)

☐ 1 No

☐ 2 Yes, window only

☐ 3 Yes, central

## Table C.1. (continued). SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

**II. QUESTIONS CONCERNING THE HEALTH OF CHILDREN 12 YEARS OF AGE OR YOUNGER WHO LIVE IN THE HOUSEHOLD**

(CARD 3)

| WRITE AT THE HEAD OF EACH COLUMN THE NAME OF EACH CHILD 12 YEARS OF AGE OR YOUNGER. THEN, IN EACH CHILD'S COLUMN CHECK ONE BOX TO ANSWER EACH OF THE FOLLOWING QUESTIONS → | (COL. 9-10) ☐☐<br>Name of 1st child | (COL. 17-18) ☐☐<br>Name of 2nd child | (COL. 25-26) ☐☐<br>Name of 3rd child | (COL. 33-34) ☐☐<br>Name of 4th child | (COL. 41-42) ☐☐<br>Name of 5th child | (COL. 49-50) ☐☐<br>Name of 6th child | (COL. 57-58) ☐☐<br>Name of 7th child | (COL. 65-66) ☐☐<br>Name of 8th child | PLEASE CHECK BOX BELOW IF THERE ARE MORE THAN 8 CHILDREN 12 YEARS OF AGE OR YOUNGER. |
|---|---|---|---|---|---|---|---|---|---|
| 1. Has your child been treated by a doctor since September 1967 for pneumonia? | (COL. 11)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 19)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 27)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 35)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 43)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 51)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 59)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 67)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 73)<br>2 |
| 2. Has your child been treated by a doctor since September 1967 for an attack of croup? | (COL. 12)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 20)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 28)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 36)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 44)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 52)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 60)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 68)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | |
| 3. Has your child been treated by a doctor since September 1967 for an attack of bronchitis, bronchiolitis, or other deep chest infection? | (COL. 13)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 21)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 29)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 37)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 45)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 53)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 61)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 69)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | |
| 4. Has your child been in the hospital since September 1967 for one of the illnesses mentioned in questions 1, 2, or 3 above? | (COL. 14)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 22)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 30)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 38)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 46)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 54)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 62)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | (COL. 70)<br>No 1<br>Yes, once 2<br>Yes, twice 3<br>Yes, more than twice 4 | |
| 5. What is the full name and address of the doctor who takes care of your child or, if you take your child to a clinic for medical care what is the name and address of the clinic? | (COL. 15-16)<br>______<br>______<br>______ | (COL. 23-24)<br>______<br>______<br>______ | (COL. 31-32)<br>______<br>______<br>______ | (COL. 39-40)<br>______<br>______<br>______ | (COL. 47-48)<br>______<br>______<br>______ | (COL. 55-56)<br>______<br>______<br>______ | (COL. 63-64)<br>______<br>______<br>______ | (COL. 71-72)<br>______<br>______<br>______ | (COL. 79-80)<br>0 3 |

## Table C.1. (continued). SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

### III. GENERAL QUESTIONS CONCERNING THE HOUSEHOLD

1. What educational level has been completed by the head of the household? (Check one box only.)

COL. 9
1 Elementary school
2 Part of high school
3 High school graduate
4 Part of college
5 College graduate
6 Graduate school
7 Other ______ (please specify)

2. What is the present employment status of the mother (or female guardian)? (Check one box only.)

COL. 10
1 Housewife or employed at home
2 Employed outside the home (full or part time)
3 Currently unemployed but usually employed outside the home.
4 Other ______ (please specify)

3. What is the race of the family?

COL. 11
1 Indian
2 Mexican-American or Spanish American
3 Negro
4 Oriental
5 White
6 Other

4. How many times have you and your family changed living quarters during the last 5 years? (Check one box only.)

COL. 12
1 Zero
2 One
3 Two
4 Three
5 Four
6 Five or more

COL. 79-80

| 10. Where did you live the longest when you were 0 to 20 years of age? (Check only one choice for each parent.) | MOTHER (or female guardian) COL. 31 | FATHER (or male guardian) COL. 32 |
|---|---|---|
| Present city, present neighborhood | 1 | 1 |
| Present city, different neighborhood | 2 | 2 |
| Different city or area of more than 200,000 people | 3 | 3 |
| Different city or area of about 50,000 to 200,000 people | 4 | 4 |
| Different city or area - A small city or town | 5 | 5 |
| Different city or area - A rural area or farm | 6 | 6 |

| 11. Where did you live the longest when you were 21 to 30 years of age? (Check only one choice for each parent.) | COL. 33 | COL. 34 |
|---|---|---|
| Not yet 21 years old | 1 | 1 |
| Present city, present neighborhood | 2 | 2 |
| Present city, different neighborhood | 3 | 3 |
| Different city or area of more than 200,000 people | 4 | 4 |
| Different city or area of about 50,000 to 200,000 people | 5 | 5 |
| Different city or area - A small city or town | 6 | 6 |
| Different city or area - A rural area or farm | 7 | 7 |

| 12. Where did you live the longest after you were 31 years of age? (Check only one choice for each parent.) | COL. 35 | COL. 36 |
|---|---|---|
| Not yet 31 years old | 1 | 1 |
| Present city, present neighborhood | 2 | 2 |
| Present city, different neighborhood | 3 | 3 |
| Different city or area of more than 200,000 people | 4 | 4 |
| Different city or area of about 50,000 to 200,000 people | 5 | 5 |
| Different city or area - A small city or town | 6 | 6 |
| Different city or area - A rural area or farm | 7 | 7 |

## Table C.1. (continued). SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

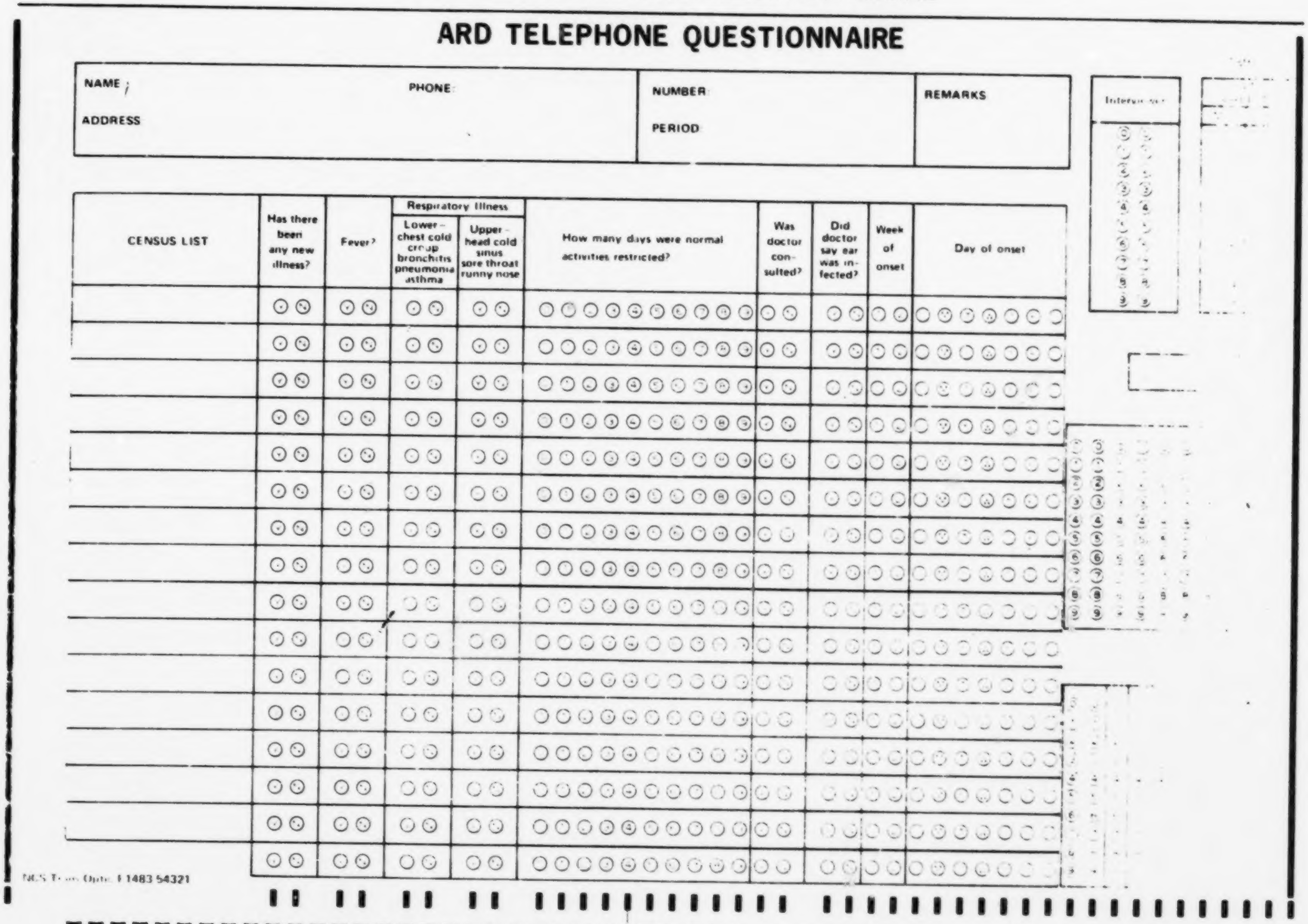
### IV. CENSUS OF HOUSEHOLD

(CARD 5-19 - ONE CARD PER PERSON)

| Names of persons living in the household | Asthma among persons living in the household (Check the appropriate box or boxes for each person.) | | | | Chronic heart or lung disease among persons living in the household (Check "yes" for each person only if chronic heart or chronic lung disease was ever diagnosed by a doctor) | | | | Sex of persons living in the household (Check below one appropriate box for each person.) | | Age of persons living in the household Complete year of birth and age for each person as indicated below | | Position in family of persons living in the household (Check below one appropriate box for each person) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Write in the column below the first and middle name of each person living in the household.) | Has this person ever had asthma diagnosed by a doctor? | | Has this asthma been active in the past two years? | | Chronic heart disease | | Chronic Lung disease | | | | | | | | | | |
| COL. 9-10 COL. 11-35 | Yes | No (COL. 36) | Yes | No (COL. 37) | Yes | No (COL. 38) | Yes | No (COL. 39) | Male | Female (COL. 40) | Year of birth | Age in years at last birthday COL. 41-42 | Head of household | Spouse COL. 43 | Child | Other | COL 79-80 |
| 01 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 05 |
| 02 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 06 |
| 03 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 07 |
| 04 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 08 |
| 05 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 09 |
| 06 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 10 |
| 07 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 11 |
| 08 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 12 |
| 09 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 13 |
| 10 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 14 |
| 11 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 15 |
| 12 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 16 |
| 13 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 17 |
| 14 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 18 |
| 15 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | 1 | 2 | ☐☐☐☐ | ☐ | 1 | 2 | 3 | 4 | 19 |

THANK YOU FOR YOUR COOPERATION

Table C.2. ACUTE RESPIRATORY DISEASE QUESTIONNAIRE



## ARD TELEPHONE QUESTIONNAIRE

NAME:
PHONE:
ADDRESS
NUMBER:
PERIOD:
REMARKS

Interviewer

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| CENSUS LIST | Has there been any new illness? | Fever? | Respiratory Illness | | How many days were normal activities restricted? | Was doctor consulted? | Did doctor say ear was infected? | Week of onset | Day of onset |
| | | | Lower-chest cold croup bronchitis pneumonia asthma | Upper-head cold sinus sore throat runny nose | | | | | |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |
| | ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ ○ ○ ○ | ○ ○ | ○ ○ | ○ ○ | ○ ○ ○ ○ ○ ○ ○ |

NCS Trans-Optic F1483 54321

## Table C.3. ASTHMA PANEL QUESTIONNAIRE

OMB No. 85-S70028

### ASTHMA PANEL QUESTIONNAIRE

(CARD 1)

I.D. # ____________________
COL (1-12)

Asthma Subject's Name ______________________________
(COL 13-44) (LAST) (FIRST) (INITIAL)

Respondent's Name ______________________________
(ENTER ONLY IF RESPONDENT IS OTHER THAN ASTHMA SUBJECT)

COL (79-80) 0 1

(CARD 2)

Subject's Address ______________________________
(COL 13-56) (HOUSE NUMBER) (STREET)

______________________________
(CITY) (STATE) (ZIP)

Subject's Telephone Number ______________________________
(COL 57-63)

Subject plans to move before next March ☐ Yes ☐ No
1 COL (64) 2

Subject agrees to participate in panel ☐ Yes ☐ No
1 COL (65) 2

Reason for refusal ______________________________ ☐☐
(COL 66-67)

Interviewer's Name ______________________________ ☐☐
(COL 68-69)

Date of Interview ☐☐☐
MO. DAY YR.
(COL 70-75)

COL. (79-80) 0 2

## Table C.3. (continued). ASTHMA PANEL QUESTIONNAIRE

(CARD 3)

### I. IDENTIFYING INFORMATION

1. Current age: ____________ COL (13-14)

2. Year of birth ____________ COL (15-18)

3. Sex: COL (19)
   Male 1 ☐
   Female 2 ☐

4. Race: COL (20)
   ☐ Indian 1
   ☐ Mexican-American or Spanish-American 2
   ☐ Negro 3
   ☐ Oriental 4
   ☐ White 5
   ☐ Other ____________ (PLEASE SPECIFY) 6

### II. ASTHMA HISTORY

| | | |
|---|---|---|
| 1. How old were you when your asthma first began? (ENTER AGE IN YEARS) | COL (21-22) ☐☐ | |
| 2. How often have you had asthma attacks within the past year? | COL (23)<br>☐ None 1<br>☐ 1–4 2<br>☐ 5–9 3<br>☐ 10 or more 4 | |
| 3. When you have an asthma attack which of the following symptoms do you usually have? | | |
| a. Shortness of breath | ☐ Yes 1 | ☐ No 2 COL (24) |
| b. Wheezing in the chest | ☐ Yes 1 | ☐ No 2 COL (25) |
| c. Fever | ☐ Yes 1 | ☐ No 2 COL (26) |
| d. Increased sputum or phlegm production | ☐ Yes 1 | ☐ No 2 COL (27) |
| 4. Do your asthma attacks ever get severe enough to keep you from school or work or from carrying on your usual activities? | ☐ Yes 1 | ☐ No 2 COL (28) |

## Table C.3. (continued). ASTHMA PANEL QUESTIONNAIRE

| | | | |
|---|---|---|---|
| 5. Do you have asthma attacks in: | | | |
| Winter (Jan. - Feb. - Mar.) ____ | ☐ Yes 1 | ☐ No 2 | COL (29) |
| Spring (Apr. - May - June) ____ | ☐ Yes 1 | ☐ No 2 | COL (30) |
| Summer (Jul. - Aug. - Sept.) ____ | ☐ Yes 1 | ☐ No 2 | COL (31) |
| Fall (Oct. - Nov. - Dec.) ____ | ☐ Yes 1 | ☐ No 2 | COL (32) |
| 6. Do many of your attacks occur in: | | | |
| Winter (Jan. - Feb. - Mar.) ____ | ☐ Yes 1 | ☐ No 2 | COL (33) |
| Spring (Apr. - May - June) ____ | ☐ Yes 1 | ☐ No 2 | COL (34) |
| Summer (Jul. - Aug. - Sept.) ____ | ☐ Yes 1 | ☐ No 2 | COL (35) |
| Fall (Oct. Nov. Dec.) ____ | ☐ Yes 1 | ☐ No 2 | COL (36) |
| No seasonal predominance ____ | ☐ Yes 1 | ☐ No 2 | COL (37) |
| 7. How many cigarettes do you usually smoke now? | CO [illegible] | | |
| Never smoked ____ | ☐ 1 | | |
| Ex-smoker ____ | ☐ 2 | | |
| Less than ½ pack per day (1–5 cigarettes) ____ | ☐ 3 | | |
| About ½ pack per day (6–14 cigarettes) ____ | ☐ 4 | | |
| About 1 pack per day (15–25 cigarettes) ____ | ☐ 5 | | |
| About 1½ packs per day (26–34 cigarettes) ____ | ☐ 6 | | |
| About 2 or more packs per day (35 or more cigarettes) ____ | ☐ 7 | | |
| 8. At your job are there fumes or dusts that tend to bring on asthma attacks? | ☐ Yes 1 | ☐ No 2 | COL (39) |
| 9. Did you ever have a skin rash or hives after taking drugs or after eating certain foods? | ☐ Yes 1 | ☐ No 2 | COL (40) |

## Table C.3. (continued). ASTHMA PANEL QUESTIONNAIRE

| | | |
|---|---|---|
| 10. Do you tend to get asthma attacks: | | |
| a. When it gets unusually cold outside? ______ | ☐ Yes 1 ☐ No 2 | COL (41) |
| b. When you get respiratory infections, like colds? ______ | ☐ Yes 1 ☐ No 2 | COL (42) |
| c. When you get upset? ______ | ☐ Yes 1 ☐ No 2 | COL (43) |
| 11. Do you have hay fever? | ☐ Yes 1 ☐ No 2 | COL (44) |
| III. GENERAL HISTORY | | |
| 1. How many rooms are there in your living quarters? Do not count bathrooms, porches, balconies, foyers, halls, or halfrooms.) | ☐ One 1<br>☐ Two 2<br>☐ Three 3<br>☐ Four 4<br>☐ Five 5<br>☐ Six 6<br>☐ Seven 7<br>☐ Eight 8<br>☐ Nine 9<br>☐ Ten 10<br>☐ Eleven 11<br>☐ Twelve or 12 more | COL (45-46) |
| 2. How many people live in your household? | ☐ One 01<br>☐ Two 02<br>☐ Three 03<br>☐ Four 04<br>☐ Five 05<br>☐ Six 06<br>☐ Seven 07<br>☐ Eight 08<br>☐ Nine 09<br>☐ Ten 10<br>☐ Eleven 11<br>☐ Twelve 12 or more | COL (47-48) |
| 3. What educational level did the head of your household complete? | ☐ Elementary school 1<br>☐ Part of high school 2<br>☐ High school graduate 3<br>☐ Part of college 4<br>☐ College graduate 5<br>☐ Graduate school 6<br>☐ Other ______ 7 (PLEASE SPECIFY) | COL (49) |
| | | COL (79-80) 0 3 |

## Table C.4. ASTHMA DIARY

Name ______________________ HEALTH DEPARTMENT ASTHMA STUDY: A

1. Please answer for each day of the week: July 25 - July 31

| | Sunday July 25 | Monday July 26 | Tuesday July 27 | Wednesday July 28 | Thursday July 29 | Friday July 30 | Saturday July 31 |
|---|---|---|---|---|---|---|---|
| Did you have an asthma episode today? | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE |

IF YOU DID HAVE AN ASTHMA EPISODE, PLEASE ANSWER THE QUESTIONS BELOW:

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| At what time did the episode begin? | ☐ MIDNIGHT TO 6 A.M.<br>☐ 6 A.M. TO NOON<br>☐ NOON TO 6 P.M.<br>☐ 6 P.M. TO MIDNIGHT | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M | ☐ 12M-6 A.M.<br>☐ 6 A.M.-12N<br>☐ 12N-6 P.M.<br>☐ 6 P.M.-12M |
| Were you in your home neighborhood when the episode began? | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES |
| Was the episode severe enough to keep you from work, school, your normal activity or from sleeping at night? | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES | ☐ NO<br>☐ YES |
| Do you feel that you can identify the cause of today's episode?<br>(If yes, please specify cause) | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ | ☐ NO<br>☐ YES<br>SPECIFY ______ |

Important: Please mail on Sunday August 1.

## Table C.5. ELDERLY PANEL QUESTIONNAIRE

---

OMB No. 86-570028

### ELDERLY PANEL QUESTIONNAIRE

I. D. # ______________________
(COL 1–12)

Subject's Name ______________________
(COL 13-44) (LAST) (FIRST) (INITIAL)

Respondent's Name ______________________
(ENTER ONLY IF RESPONDENT IS OTHER THAN SUBJECT)

COL (79-80) 0 1

---

(CARD 2)

Subject's Address ______________________
COL (13-56) (HOUSE NUMBER) (STREET)

______________________
(CITY) (STATE) (ZIP)

Subject's telephone number ______________________
(COL 57-63)

Subject plans to move before next March: ☐ Yes 1 ☐ No 2 COL (64)

Subject agrees to participate in panel: ☐ Yes 1 ☐ No 2 COL (65)

Reason for refusal: ______________________
(COL 66-67)

Interviewer's Name: ______________________
(COL 68-69)

Date of Interview: ☐☐☐
MO. DAY YR.
COL (70-73)

COL (79-80) 0 2

## Table C.5. (continued). ELDERLY PANEL QUESTIONNAIRE

(CARD 3)
IDENTIFYING INFORMATION

(CARD 3)

IDENTIFYING INFORMATION

1. Current age: ______________________ COL (13-14)

2. Year of birth: ______________________ COL (15-16)

3. Sex: COL (17)
☐ Male 1
☐ Female 2

COL (18)
4. Race
☐ Indian 1
☐ Mexican-American or Spanish-American 2
☐ Negro 3
☐ Oriental 4
☐ White 5
☐ Other 6 (PLEASE SPECIFY)

| 5. How many cigarettes do you usually smoke now? | COL (19) |
|---|---|
| Never smoked ______ | ☐ 1 |
| Ex-smoker ______ | ☐ 2 |
| Less than ½ pack per day (1-5 cigarettes) ______ | ☐ 3 |
| About ½ pack per day (6-14 cigarettes) ______ | ☐ 4 |
| About 1 pack per day (15-25 cigarettes) ______ | ☐ 5 |
| About 1½ packs per day (26-34 cigarettes) ______ | ☐ 6 |
| About 2 or more packs per day (35 or more cigarettes) ______ | ☐ 7 |

6. At your job, are you now or have you been frequently exposed to irritating smoke, dust, or fumes? (Do not include neighborhood or home exposures.) (COL 20) ☐ Yes 1 ☐ No 2

IF YOU ANSWERED "NO" TO QUESTION 6, SKIP THE NEXT THREE QUESTIONS BELOW.

6a. If the answer to question 6 is "yes", what kind of irritant were you exposed to? (For example: coal dust, cutting oils, asbestos, mine dust, smelter fumes, raw cotton dust.) ______________________
______________________

## Table C.5. (continued). ELDERLY PANEL QUESTIONNAIRE

| | |
|---|---|
| 6b. If the answer to question 6 is "yes", what kind of work did you perform in this job? (For example: miner, maintenance, assembly line, supervisor.) | ________________<br>________________ |
| 6c. If the answer to question 6 is "yes", how long were you exposed? | (COL 21) |
| Less than 1 year | ☐ 1 |
| 1 to 5 years | ☐ 2 |
| 6 to 10 years | ☐ 3 |
| More than 10 years | ☐ 4 |
| III. GENERAL HISTORY<br>1. How many rooms are there in your living quarters? Do not count bathrooms, porches, balconies, foyers, halls, or halfrooms.) | (COL 22 23)<br>1 ☐ One<br>2 ☐ Two<br>3 ☐ Three<br>4 ☐ Four<br>5 ☐ Five<br>6 ☐ Six<br>7 ☐ Seven<br>8 ☐ Eight<br>9 ☐ Nine<br>10 ☐ Ten<br>11 ☐ Eleven<br>12 ☐ Twelve or more |
| 8. How many people live in your household? | (COL 24 25)<br>☐ One 01<br>☐ Two 02<br>☐ Three 03<br>☐ Four 04<br>☐ Five 05<br>☐ Six 06<br>☐ Seven 07<br>☐ Eight 08<br>☐ Nine 09<br>☐ Ten 10<br>☐ Eleven 11<br>☐ Twelve or more 12 |
| 9. What educational level did the head of your household complete? | COL (26)<br>☐ Elementary school 1<br>☐ Part of high school 2<br>☐ High School graduate 3<br>☐ Part of college 4<br>☐ College graduate 5<br>☐ Graduate school 6<br>☐ Other __________ 7 (PLEASE SPECIFY)<br>COL (79 80) 0 3 |

## Table C.6. ELDERLY PANEL HEALTH QUESTIONNAIRE

FORM APPROVED
Budget Bureau
No. 85 - R0133

### HEALTH QUESTIONNAIRE

S

Study # ____________________ (COL 1 - 12)

I. Name ____________________ (LAST) (COL 13 - 33) (FIRST) (M.I.)

Address ____________________ (COL 34 - 77)

(COL 79 - 80) 0 1

| | | | | |
|---|---|---|---|---|
| II. | 1. Do you usually cough first thing in the morning in winter? | Yes ☐ 1 | No ☐ 2 | (COL 13) |
| | 2. Do you usually cough during the day or night in winter? | Yes ☐ 1 | No ☐ 2 | (COL 14) |
| | *If you answered "Yes" to question 1 or 2, please answer question 3.* | | | |
| | 3. Do you cough like this on most days or nights for as much as three months each year? | Yes ☐ 1 | No ☐ 2 | (COL 15) |
| | 4. Do you usually bring up phlegm from your chest first thing on getting up in the morning? | Yes ☐ 1 | No ☐ 2 | (COL 16) |
| | 5. Do you usually bring up phlegm from your chest during the day or at night in winter? | Yes ☐ 1 | No ☐ 2 | (COL 17) |
| | *If you answered "Yes" to question 4 or 5, please answer question 6.* | | | |
| | 6. Do you bring up phlegm like this on most days for as much as three months each year? | Yes ☐ 1 | No ☐ 2 | (COL 18) |
| | 7. Have you ever had heart trouble? | Yes ☐ 1 | No ☐ 2 | (COL 19) |
| | 8. Have you ever had pain or discomfort in your chest brought on by exertion | Yes ☐ 1 | No ☐ 2 | (COL 20) |
| | 9. Have you ever been told by your doctor that you had angina, or angina pectoris? | Yes ☐ 1 | No ☐ 2 | (COL 21) |
| | 10. Have you ever been told by your doctor that your heart was large? | Yes ☐ 1 | No ☐ 2 | (COL 22) |
| | 11. Do you get short of breath walking with other people at an ordinary pace on the level? | Yes ☐ 1 | No ☐ 2 | (COL 23) |
| | 12. Have your legs ever become swollen? | Yes ☐ 1 | No ☐ 2 | (COL 24) |
| | *If so, what was the cause of the swelling?* Heart trouble<br>Lung trouble<br>Kidney trouble<br>Varicose veins<br>Other | ☐ 1<br>☐ 2<br>☐ 3<br>☐ 4<br>☐ 5 | | (COL 25) |
| | 13. Have you ever had a heart attack? | Yes ☐ 1 | No ☐ 2 | (COL 26) |
| | 14. Do you take any medicine for your heart or to help you lose water? | Yes ☐ 1 | No ☐ 2 | (COL 27) |

**Table C.6. (continued). ELDERLY PANEL HEALTH QUESTIONNAIRE**

15. How old are you? ________ Years (COL 28 - 29)

16. What is your sex? Male ☐ 1 Female ☐ 2 (COL 30)

17. How many years have you lived at your present address? ________ Years (COL 31 - 32)

18. How many years have you lived in your present borough or county? ________ Years (COL 33 - 34)

19. What is your home telephone number ________ (COL 35 - 41)

Thank you very much for your cooperation.

## Table C.7. "WELL" PANEL DIARY

BOB #85-R0133

NAME ______________________

HEALTH DEPARTMENT STUDY: 0

PLEASE ANSWER UNLY ONCE FOR THE ENTIRE WEEK.

1. DID YOU HAVE A NEW COUGH, COLD OR SORE THROAT DURING THE WEEK? ☐ NO ☐ YES
2. HAVE YOU VISITED A DOCTOR THIS WEEK BECAUSE YOU WERE FEELING ILL? ☐ NO ☐ YES
3. WERE YOU ADMITTED TO A HOSPITAL THIS WEEK? ☐ NO ☐ YES

IN THE PAST WEEK HAVE YOU HAD ANY OF THE FOLLOWING SYMPTOMS:

COUGH, CHEST PAINS, WHEEZING IN THE CHEST, PHLEGM FROM THE CHEST? ☐ NO ☐ YES

(IF YES, PLEASE CHECK THE DAY OF THE WEEK YOU HAD THE SYMPTOMS)

| | SUN. | MON. | TUES. | WED. | THUR. | FRI. | SAT. |
|---|---|---|---|---|---|---|---|
| CHEST PAINS | | | | | | | |
| WHEEZING | | | | | | | |
| PHLEGM | | | | | | | |
| COUGH | | | | | | | |

IMPORTANT: PLEASE MAIL ON SUNDAY

## Table C.8. "HEART" PANEL DIARY

NAME ____________________

OMB #85-R0133

HEALTH DEPARTMENT STUDY: I

PLEASE ANSWER QUESTIONS 1, 2 & 3 FOR EACH DAY OF THE WEEK.

| | SUNDAY | MONDAY | TUESDAY | WEDNESDAY | THURSDAY | FRIDAY | SATURDAY |
|---|---|---|---|---|---|---|---|
| 1. Is your shortness of breath today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |
| 2. Have you had any attacks of angina (dull chest pain) during the past 24 hours? | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE |
| 3. Is the swelling in your feet today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |

II. PLEASE ANSWER QUESTIONS 4, 5, & 6 ONCE FOR THE ENTIRE WEEK.

4. Did you have a new cough, cold, or sore throat during the week? ☐ NO ☐ YES
5. Have you visited a doctor this week because you were ill? ☐ NO ☐ YES
6. Were you admitted to a hospital this week? ☐ NO ☐ YES

IMPORTANT: PLEASE MAIL ON SUNDAY

## Table C.9. "LUNG" PANEL DIARY

NAME ____________________ HEALTH DEPARTMENT STUDY: 2 BOB [illegible]

I. PLEASE ANSWER QUESTIONS 1, 2 & 3 FOR EACH DAY OF THE WEEK.

| | SUNDAY | MONDAY | TUESDAY | WEDNESDAY | THURSDAY | FRIDAY | SATURDAY |
|---|---|---|---|---|---|---|---|
| 1. Is your cough or phlegm production today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |
| 2. Is the wheezing in your chest today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |
| 3. Is your shortness of breath today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |

II. PLEASE ANSWER QUESTIONS 4, 5, & 6 ONCE FOR THE ENTIRE WEEK.

4. Did you have a new cough, cold, or sore throat during the week? ☐ NO ☐ YES
5. Have you visited a doctor this week because you were ill? ☐ NO ☐ YES
6. Were you admitted to a hospital this week? ☐ NO ☐ YES

**IMPORTANT: PLEASE MAIL ON SUNDAY**

## Table C.10. "HEART AND LUNG" PANEL DIARY

BO #85-R0133

NAME ____________________

HEALTH DEPARTMENT STUDY: 3

I. PLEASE ANSWER QUESTIONS 1, 2 & 3 FOR EACH DAY OF THE WEEK.
& 4

| | SUNDAY | MONDAY | TUESDAY | WEDNESDAY | THURSDAY | FRIDAY | SATURDAY |
|---|---|---|---|---|---|---|---|
| 1. Is your shortness of breath today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |
| 2. Have you had any attacks of angina (dull chest pain) during the past 24 hours? | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE | ☐ NO<br>☐ YES, ONE<br>☐ YES, MORE THAN ONE |
| 3. Is the swelling in your feet today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |
| 4. Is your cough or phlegm production today much worse, worse, the same, or better than usual? | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT | ☐ MUCH WORSE<br>☐ WORSE<br>☐ SAME<br>☐ BETTER<br>☐ NEVER PRESENT |

II. PLEASE ANSWER QUESTIONS 5, 6 & 7 ONCE FOR THE ENTIRE WEEK.

5. Did you have a new cough, cold, or sore throat during the week? ☐ NO ☐ YES

6. Have you visited a doctor this week because you were ill? ☐ NO ☐ YES

7. Were you admitted to a hospital this week? ☐ NO ☐ YES

IMPORTANT: PLEASE MAIL ON SUNDAY

# APPENDIX D:
# ABBREVIATIONS AND CONVERSION FACTORS

## ABBREVIATIONS

| | |
|---|---|
| °C | degrees Celsius (Centigrade) |
| CAMP | Continuous Air Monitoring Program |
| CHESS | Community Health and Environmental Surveillance System |
| COH | coefficient of haze |
| cm | centimeters |
| EDTA | ethylenediamine tetraacetic acid |
| EPA | U.S. Environmental Protection Agency |
| °F | degrees Fahrenheit |
| $FEV_{0.75}$ | 0.75-second forced expiratory volume |
| g | grams |
| Hg | mercury |
| LMB | lower-middle class black |
| LMW | lower-middle class white |
| LRI | lower respiratory illness |
| m | meters |
| *M* | molar |
| max. | maximum |
| MBTH | 3-methyl-2-benzothiazolone hydrozone hydrochloride |
| mg | milligrams |
| min | minutes |
| min. | minimum |
| ml | milliliters |
| mm | millimeters |
| mo | months |
| mph | miles per hour |
| *N* | normal |
| NASN | National Air Sampling Network |
| NDEA | N-1-naphthylethylenediamine dihydrochloride |
| nm | nanometers |
| NO | nitric oxide |
| $NO_2$ | nitrogen dioxide |
| NYC-DAR | New York City Department of Air Resources |
| p | probability |
| ppm | parts per million |
| PRA | pararosaniline hydrochloride |
| RSP | respirable suspended particulates |
| sec | seconds |
| SES | socioeconomic status |
| SN | suspended nitrates |
| $SO_2$ | sulfur dioxide |
| SS | suspended sulfates |
| TCM | sodium tetrachloromercurate |
| $T_{min}$ | minimum temperature |
| TSP | total suspended particulate |
| UMW | upper-middle class white |
| $X^2$ | Chi Square |
| yr | years |
| μg | micrograms |
| μm | micrometers |

## CONVERSION FACTORS

Primary data, such as pollutant concentrations, in this report are given in metric units. For convenience some intermediate data, such as emission rates, used in the calculations are given in nonmetric units as originally reported. If desired, metric equivalents for those data can be obtained by application of the conversion factors given below.

| *Multiply* | *By* | *To obtain* |
|---|---|---|
| cubic feet | $2.83 \times 10^{-2}$ | cubic meters |
| degrees Fahrenheit* | 5/9 | degrees Celsius (Centigrade) |
| feet | $3.05 \times 10^{-1}$ | meters |
| inches | $2.54 \times 10^{-2}$ | meters |
| miles | $1.61 \times 10^{3}$ | meters |
| tons | $1.02 \times 10^{3}$ | kilograms |

***To obtain Celsius (C) temperature readings from Fahrenheit (F) readings, use the following formula: $C = (5/9)(F - 32)$.**

# APPENDIX E: BIBLIOGRAPHY

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